Chapter 18: Anxiety Disorders

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Introduction

As a group, anxiety disorders are the most common mental disorders. The lifetime prevalence of anxiety disorders in various countries has been found to range between 9.2% and 28.7%, with the best-estimate lifetime prevalence rate for all anxiety disorders calculated at 16.6% (Somers et al., 2006).

The main feature of anxiety disorders is the presence of pathological anxiety, but emotions such as disgust, irritability and shame may also be prominent. Pathological anxiety differs from normal anxiety in a number of ways (Table 18.1). Individuals with anxiety disorders usually have a preserved insight that their anxiety is excessive and/or unreasonable.

Fear is closely related to anxiety. Both appear in response to a perceived threat and are often accompanied by similar bodily sensations, i.e., symptoms of autonomic nervous system hyperarousal (see Figure 18.1). However, there are also differences. While the threat in fear is easier to identify, external to the individual, more specific and immediate, the threat in anxiety tends to be elusive, internal, diffuse and lingering. The onset of fear is often abrupt and its course is acute or episodic, whereas the onset of anxiety is more often vague and its course is usually chronic. In contrast to fear where a typical response to threat is immediate "fight or

flight", the typical response in anxiety is vigilance. Most anxiety disorders have features of both fear and anxiety.

Table 18.1. Differences between normal and pathological anxiety.

	Normal anxiety	**Pathological anxiety**
Intensity	Minimal to mild	Moderate to severe
Duration	Short-lasting	Persistent
Quality of the experience	Understandable, rational	Puzzling, overwhelming, with little or no control over the anxiety
Sense of danger	Realistic	Exaggerated
Significant distress or impairment in functioning	No	Yes

Several conditions are classified in the group of anxiety disorders: panic disorder, agoraphobia, social anxiety disorder (social phobia), specific phobia and generalised anxiety disorder (GAD). The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR) (American Psychiatric Association, 2022) also includes separation anxiety disorder and selective mutism, conditions that are usually encountered in childhood, although 40% of adults with separation anxiety disorder have been reported to experience an onset after the age of 18 years (Silove et al., 2015). If pathological anxiety is caused by a substance or a medical condition, such disorder is diagnosed as a substance-induced anxiety disorder or anxiety disorder due to another medical condition, respectively. Although obsessive-compulsive disorder and posttraumatic stress disorder are related to the anxiety disorders, in the DSM-5-TR each has been classified in its own category (American Psychiatric Association, 2022). Panic attacks are described below, followed by a brief outline of each anxiety disorder and treatment considerations.

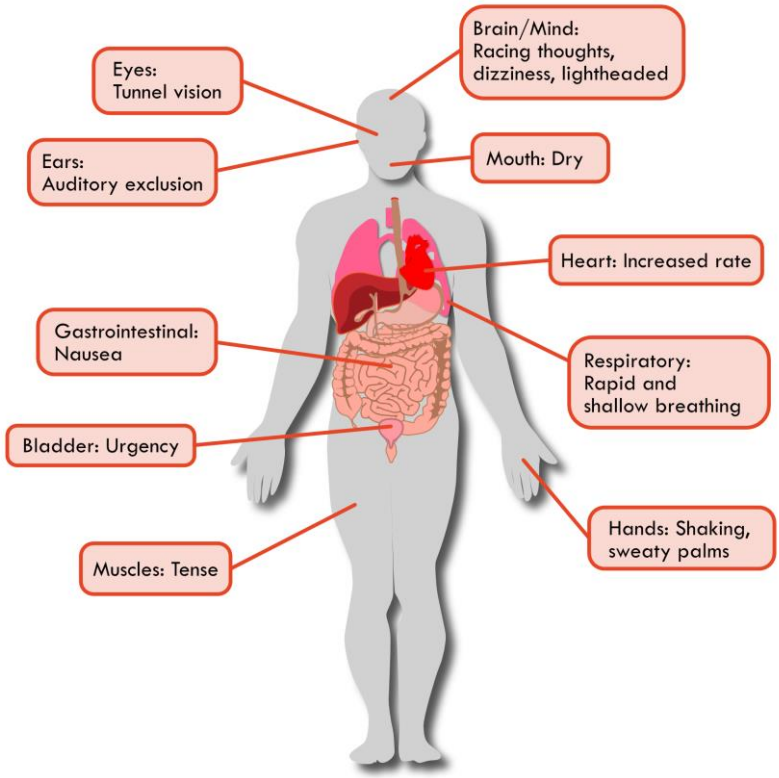

Figure 18.1. Physiological responses to anxiety.

Panic Attack

Panic attacks are characterised by an abrupt onset and rapid escalation of physical symptoms of anxiety, accompanied by intense fear. The numerous and distressing bodily symptoms are for the most part a consequence of autonomic nervous system hyperarousal. The peak of a panic attack is reached within minutes and attacks usually do not last very long – rarely more than 30 minutes.

The very first panic attack is often experienced as "out of the blue"; a similar experience may be reported for a few panic attacks occurring initially. Subsequent panic attacks are more likely to have cognitive (e.g., anxious thoughts), somatic (e.g., feeling hot, increased heart rate) or situational triggers, although these may be subtle and not obvious to the individual. Eventually, people usually learn to associate panic attacks with certain situations, and the attacks tend subsequently to be

anticipated and felt to be more likely in these situations. It is important to note that panic attacks may occur in any disorder: they are not pathognomonic of panic disorder.

Panic Disorder

Panic disorder comprises two components: recurrent panic attacks and anticipatory anxiety. Panic attacks in panic disorder are associated with intense fears of physical or mental catastrophe perceived as imminent. Such catastrophe includes dying, losing consciousness and passing out, losing control or "going crazy".

Lifetime prevalence of panic attacks
- Up to 28.7%

Lifetime prevalence of panic disorder
- From 0.4% to 3.8%
- Best-estimate: 1.2% (Somers et al., 2006)

Male-to-female ratio
- 1:2-3

Usual onset of panic disorder
- Third decade of life

Help-seeking patterns
- Usually quick help-seeking due to intense and frightening physical symptoms and the accompanying fear
- Help is often sought in hospital emergency departments or primary care and specialised medical settings

Anticipatory anxiety is a key feature of panic disorder and represents a distressing and persistent fear of another panic attack. The essence of this fear is anticipation of the (unlikely) consequences of panic attacks, such as death, heart attack or other bodily catastrophe, loss of control, psychotic breakdown or humiliation. Anticipatory anxiety may lead to changes in behaviour, usually in the form of avoidance of the activities or situations (e.g., exercising or crowded shopping centres) that could trigger a panic attack or some of its symptoms. Epidemiological and help-seeking characteristics of panic disorder are presented above. In addition to the indirect costs of functional impairment and disability, panic disorder is associated with the direct costs of frequent visits to medical practitioners and numerous medical investigations.

Little is known about the aetiology of panic disorder. A genetic predisposition has been suggested, but it is unclear what constitutes a genetic vulnerability to panic disorder and how specific it is for this condition. A relatively specific risk factor for panic disorder is a high level of anxiety sensitivity (Schmidt et al., 1997), which denotes a fear of anxiety and its bodily symptoms based on beliefs about their dangerousness. The most commonly invoked psychological mechanism in panic attacks is catastrophic misinterpretation of bodily sensations and symptoms (Clark, 1986). In terms of neurobiology, an abnormally sensitive anxiety-regulating mechanism originating in the amygdala may be involved in all anxiety disorders. The frequent predominance of respiratory symptoms during panic attacks has been hypothesised to be due to hyperventilation, hypersensitivity to carbon dioxide of the brain-stem chemoreceptors or lower threshold for activating the suffocation alarm mechanism. Panic attacks may occur as a consequence of an increased central noradrenergic activity, with hypersensitivity of the presynaptic α-2 receptors and increased firing rate of the locus coeruleus. An additional factor may be a failure of the gamma-aminobutyric acid (GABA) neurotransmitter system to inhibit the locus coeruleus. Abnormalities in the serotonin neurotransmitter systems have also been implicated in the aetiology of panic disorder.

The diagnosis of panic disorder is based on clinical presentation and exclusion of the relevant medical conditions that may cause panic attacks and/or high levels of anxiety (Table 18.2). Panic attacks may occur in the course of other mental disorders and in such cases, an additional diagnosis of panic disorder may or may not be warranted. Substance misuse, other anxiety disorders and depression frequently accompany panic disorder.

The course of panic disorder varies from one person to another. Agoraphobia is a common complication. At least one third of patients recover, but as many as 50% report a chronic, fluctuating course, with remissions and exacerbations. The severity of panic disorder tends to decrease with ageing. Studies have established links between panic disorder and a higher risk of suicide attempts, suicide and cardiovascular disease, but these relationships are poorly understood.

Table 18.2. Medical conditions that may cause panic attacks and/or high levels of anxiety.

Frequent association with panic attacks	
Endocrine and metabolic disturbances	• Hyperthyroidism • Hypoglycaemia
Cardiac conditions	• Cardiac arrhythmias (e.g., paroxysmal atrial tachycardia) • Mitral valve prolapse
Neurological and vestibular conditions	• Complex partial (temporal, psychomotor) epilepsy • Vestibular dysfunction
Less common association with panic attacks	
Endocrine and metabolic disturbances	• Hypothyroidism • Hypoparathyroidism • Hyperparathyroidism • Cushing's syndrome • Menopause-associated endocrine disturbances • Electrolyte disturbances • Pheochromocytoma
Pulmonary conditions	• Pulmonary embolus
Gastrointestinal disorders	• Irritable bowel syndrome

Agoraphobia

Agoraphobia is a fear of multiple, interrelated situations from which it might be difficult or impossible to escape immediately (e.g., being in crowded or enclosed places such as shopping centres, cinemas, airplanes and tunnels or using public transport such as buses and trains), those in which the person is alone or outside of their safety zone so that immediate medical or other help might not be available (e.g., travelling far away from home, staying at home alone) and situations from which it might be awkward or embarrassing to escape immediately (e.g., standing in line, sitting in the middle of a row in a theatre). These situations are typically avoided. Avoidance may be of such proportions that the person becomes homebound and quite incapacitated. Individuals with agoraphobia often rely on a "phobic companion" (usually a partner or family member) to accompany them to feared situations.

Agoraphobic situations are frequently feared because of the expectation that panic attacks and symptoms would occur in these situations. Therefore, prevention of panic attacks and symptoms is the usual aim of avoidance in agoraphobia. This also explains a strong relationship between agoraphobia and panic disorder and their frequent co-occurrence. However, agoraphobia can also develop independently from panic, for example in the context of the fears of falling, being incontinent, getting lost, having an accident or being mugged.

Epidemiological and help-seeking characteristics of agoraphobia are shown below. Agoraphobia is associated with significant functional impairment, especially when the person is homebound.

Lifetime prevalence

- From 0.7% to 10.8%
- Best-estimate: 3.1% (Somers et al., 2006)

Male-to-female ratio

- 1:3-4

Usual onset

- Late adolescence or early adulthood

Help-seeking patterns

- Help-seeking depends on the extent of avoidance and the associated functional impairment
- Help is often sought from general practitioners or mental health services

The agoraphobic fear is usually explained as a consequence of learning, i.e., of associating unpleasant anxiety symptoms, panic attacks, with certain situations. These situations are subsequently avoided, with avoidance reinforcing the agoraphobic fear.

The diagnosis of agoraphobia is based on the fear and avoidance of a number of typical situations, as described above. A proper assessment includes ascertaining the specific fears linked to avoided situations and the extent of avoidance. It is important to distinguish agoraphobia from certain types of specific phobia (especially claustrophobia) and depressive disorders.

Agoraphobia is a chronic condition, with little or no spontaneous improvement. The extent of disability and prognosis may depend on the availability

of a phobic companion and other personal circumstances. If agoraphobia is aetiologically related to panic attacks, successful treatment of the latter may alleviate, but not necessarily eliminate, agoraphobic fear and avoidance. The most important long-term complications of agoraphobia are depression and alcohol misuse.

Social Anxiety Disorder (Social Phobia)

Social anxiety disorder is defined as a prominent and persistent fear of situations in which the person is or might be exposed to the scrutiny of others. These include both performance-type situations (giving a talk or doing something else in front of others) and various interpersonal interactions (ranging from simple conversations with unfamiliar people and asking strangers for directions to the more challenging situations involving communication with authority figures or expressing disagreement). Avoidance of social situations is the usual way of coping with social anxiety. If avoidance is not possible, there is endurance of social situations with much distress. In milder forms of social anxiety disorder, only performance-type situations tend to be feared and avoided.

The key phenomenon in social anxiety disorder is fear of negative evaluation, which is evident in concerns about coming across as "stupid", abnormal or incompetent and the humiliation that the person fears will result. Fear of having noticeable anxiety-related bodily symptoms such as blushing, sweating or trembling, is also common. Social anxiety disorder is closely related to shyness, defined as the tendency to feel awkward, worried or tense during social encounters. However, the former denotes higher levels of anxiety, greater avoidance of social situations and decreased ability to adapt to social situations and respond to positive social cues. Unlike shy individuals, those with social anxiety disorder remain very anxious throughout their exposure to a social situation and despite any positive feedback or encouragement that they receive during such exposure.

Epidemiological and help-seeking characteristics of social anxiety disorder are shown below. Multiple areas of functioning are adversely affected. Severely affected individuals, about 30% of those with social anxiety disorder, may never form relationships, their educational achievement is not commensurate with their intellectual abilities and their participation in the workforce may only be marginal. A greater number of feared social situations is associated with greater severity and disability. Of all the anxiety disorders, long-term disability was reported to be most prominent in social anxiety disorder (Hendriks et al., 2016).

The aetiology of social anxiety disorder involves a number of biological and psychological factors. A genetic predisposition has been demonstrated, with most of

the vulnerability shared with other anxiety disorders and depression. A relatively specific risk factor is behavioural inhibition to the unfamiliar (Kagan et al., 1984), which can be observed very early in life as increased anxiety in response to strangers, increased startle response and difficulty settling after novel stimuli. A cognitive model (Rapee and Heimberg, 1997) postulates that social anxiety disorder results from the negative assumptions and beliefs about oneself, others and social situations (e.g., "I am inadequate, others are judgemental and social situations are a testing ground"). These assumptions and beliefs lead to appraisals of social situations as threatening and are maintained through avoidance of these situations, use of safety behaviours and multiple biases in information processing.

Lifetime prevalence

- From 0.5% to 16%
- Best-estimate: 3.6% (Somers et al., 2006)

Male-to-female ratio

- 1:1.5-2

Usual onset

- Early adolescence

Help-seeking patterns

- Delayed help-seeking, usually due to shame and embarrassment or lack of realisation that the condition can be treated
- When help is sought, this is often for complications such as depression or substance use disorder or because of an important life change (e.g., going to a different school or starting a new job)

The diagnosis of social anxiety disorder is based on the presence of severe anxiety in multiple social situations, which is usually accompanied by extensive avoidance. At the time of assessment, it is important to ascertain the presence of complications, particularly depression and substance use disorder. Due to its availability and anxiolytic and disinhibiting properties, alcohol is commonly misused by individuals with social anxiety disorder. While there is much overlap between social anxiety disorder and avoidant personality disorder, they seem to be distinct conditions (Lampe and Malhi, 2018).

The course of social anxiety disorder is chronic. Fluctuations in the intensity of social anxiety do occur, but without treatment, individuals with the condition typically remain impaired in many areas of functioning.

Specific Phobia

Various types of phobic disorders are subsumed under the diagnostic entity of specific phobia. They include phobia of animals, blood-injection-injury phobia (i.e., fear of the sight of blood, injured tissues or needle penetrating the skin), situational phobia (i.e., fears of driving and flying and claustrophobia), phobias of water, heights and storms, dental phobia and phobia of choking or vomiting. Individuals with specific phobia experience intense fear in the presence of their "phobic stimulus", i.e., the specific object, situation, activity or natural phenomenon that they are afraid of. A fearful response can have the intensity and other characteristics of a panic attack. The fear is based on the perception of threat posed by the phobic stimulus. For example, a person with claustrophobia is typically afraid of being unable to breathe in a small, enclosed space. Some types of specific phobia, especially phobia of insects and blood-injection-injury phobia, are related to intense and distressing disgust responses. Blood-injection-injury phobia is unique among the phobias because it is as common in males as it is in females and is often characterised by a vasovagal reaction, with bradycardia, hypotension and fainting rather than the symptoms of autonomic hyperarousal generally seen in panic attacks.

Avoidance is the usual way of coping with fear in specific phobia. It is generally easier and less costly to avoid the circumscribed phobic stimuli in specific phobia than it is to avoid multiple agoraphobic situations in agoraphobia and social situations in social anxiety disorder. This explains the fact that individuals with specific phobia are usually less disabled by their fears and avoidance than those with agoraphobia and social anxiety disorder and that they are encountered less often in clinical settings. Epidemiological and help-seeking characteristics of specific phobia are shown below.

A genetic predisposition to specific phobia is likely to be nonspecific. Learning theories have provided a useful explanatory framework for specific phobia. Thus, phobia may be learned through a traumatic conditioning (direct aversive experience with the phobic stimulus), vicariously (observation of the fear in others) or via transmission of the information on the dangerousness of certain objects or situations. Some types of specific phobia (phobia of heights, water and spiders) have been conceptualised as "innate" fears, which may have a survival value and do not have to be learned (Menzies and Clarke, 1995).

The diagnosis of specific phobia requires the presence of the characteristic fear and avoidance and is warranted only if the fear is persistent and has caused significant distress or functional impairment. The fear is out of proportion to the actual threat posed by the phobic stimuli and to the sociocultural context. Insight that the fear is irrational or excessive is usually preserved, but may be absent in

children. In clinical practice, specific phobia may need to be distinguished from agoraphobia.

Specific phobia is typically a chronic disorder and fears rarely abate. The impairment is directly related to the degree of avoidance. Specific phobia may precede various other anxiety disorders. The prognosis is generally good, especially with treatment.

Lifetime prevalence

- From 0.6% to 12.5%
- Best-estimate: 5.3% (Somers et al., 2006)

Male-to-female ratio

- 1:2 (except for blood-injection-injury phobia)

Usual onset

- Childhood (except for situational phobia which tends to have an onset in adolescence or early adulthood)

Help-seeking patterns

- Delayed help-seeking, usually due to successful avoidance and relatively low functional impairment
- When help is sought, this is often due to changes in life circumstances that prevent ongoing avoidance (e.g., commencing a job that involves frequent air travel makes avoidance of flying no longer possible)

Generalised Anxiety Disorder (GAD)

The main characteristics of GAD are pathological worry and symptoms of tension. Pathological worry is a key symptom and GAD does not refer only to high levels of anxiety. Symptoms of autonomic hyperarousal may also be present, but they are usually less severe than in other anxiety disorders. Individuals with GAD worry about a range of issues, including health, relationships, family, work and finances. Pathological worry differs from normal worry in terms of the anxiety-amplifying and uncontrollable "what if" pattern of thinking. Such worry denotes an almost incessant and fruitless overthinking, whereby the threat is grossly exaggerated, focusing on the task to find a rational solution is diminished, further problems continue to be anticipated and there is no closure in sight. Pathological worry thus interferes with problem solving and decision making, which causes impairment in various domains of functioning.

Tension in GAD is usually experienced or observed as nervousness, feeling "keyed up" or "on edge", irritability, exaggerated startle response, hypervigilance, restlessness or inability to relax. Muscle tension is one of the characteristic symptoms of GAD, with common complaints of tension headache and stiffness or pain in the neck, shoulder or back. Muscle spasms, tic-like movements, jerks, fine tremor and difficulty swallowing may also be present. Many individuals with GAD complain of sleep disturbance and fatigue, largely as a consequence of tension and constant and unproductive worrying.

Generalised anxiety disorder is arguably the most common anxiety disorder in the general population. Epidemiological and help-seeking characteristics of GAD are shown in the box below.

Various biological and psychological factors are likely to play a role in the aetiology of GAD. A genetic predisposition to GAD is so similar to the genetic predisposition to major depressive disorder that the two conditions have been regarded as genetically indistinguishable (Kendler et al., 1992). Various neurobiological mechanisms (e.g., hyperactivity of the noradrenaline system and a decreased function of the GABA-A receptors) have also been implicated, suggesting that GAD is a heterogeneous condition. Psychological models of GAD have mainly focused on the origins and functions of pathological worry. Thus, it has been hypothesised that pathological worry is largely driven by a difficulty in coming to terms with uncertainty (intolerance of uncertainty) (Ladouceur et al., 1997), with a notion that worrying could perhaps cease only with attainment of complete certainty. Another theory postulates that worrying allows avoidance of unpleasant bodily symptoms that accompany strong emotional states (Borkovec et al., 1998). Beliefs about the benefits of worrying (e.g., that worrying can prevent catastrophic outcomes) may reinforce pathological worry and thereby help maintain GAD (Freeston et al., 1994).

Making the diagnosis of GAD may not be easy. Patients rarely present with pathological worry as their main problem because many have reluctantly accepted it as a part of their personality that cannot be changed. Instead, a large proportion of patients present with symptoms such as insomnia or headaches, or vague complaints of "stress", especially when seeking help from general practitioners. By the time they seek help, many individuals with GAD have become depressed, and GAD may be overshadowed by depressive symptoms. For these reasons, many cases of GAD are missed or misdiagnosed, GAD is rarely seen alone in clinical settings and is often diagnosed as a condition accompanying other mental disorders, usually major depressive disorder or another anxiety disorder.

The course of GAD is chronic. Besides depression, substance use disorders can also complicate its course. The impairment associated with GAD can be substantial.

Lifetime prevalence
- From 1.9% to 31.1%
- Best-estimate: 6.2% (Somers et al., 2006)

Prevalence across life-span
- High prevalence in all age groups
- Most common anxiety disorder among the elderly

Male-to-female ratio
- 1:2

Onset
- Usually in late adolescence or early adulthood
- Onset can occur at any age

Help-seeking patterns
- Delayed help-seeking, often due to perception of worrying and chronic anxiety as part of one's "normal" personality
- When help is sought, this is often due to complications such as depression

Treatment of Anxiety Disorders

The treatment approach to anxiety disorders is based on several components: 1) A thorough biopsychosocial and lifestyle assessment; 2) Exclusion of the relevant medical conditions and psychotropic substances as the causes of pathological anxiety; 3) Identification of the factors that maintain the disorder; 4) Collaboration between mental health professionals.

Treatment of the various anxiety disorders has many commonalities, and the same or similar treatment modalities are used across the anxiety disorders. The goals of treatment include a substantial decrease in anxiety and anxiety-related behaviours (e.g., avoidance), better coping with anxiety, decrease in vulnerability, prevention of recurrences and complications and improved functioning and quality of life. Treatments differ in terms of their potential to help patients reach these goals and how they go about reducing the impact of pathological anxiety.

Pharmacotherapy and cognitive behaviour therapy (CBT) have been most widely used and their efficacy has been demonstrated by numerous controlled trials (see Chapter 33). Modifications of CBT, such as mindfulness-based therapy and acceptance and commitment therapy, have also shown efficacy. In addition, several controlled studies have demonstrated the efficacy of psychodynamic psychotherapy. Pharmacotherapy and psychological treatment are often combined in clinical practice and there is some evidence to support this approach.

Pharmacotherapy

Pharmacological agents are used to alleviate distress and symptoms of anxiety and any co-occurring disorders, especially depression (see Chapter 35). Medications such as benzodiazepines are particularly effective in giving rapid relief of acute anxiety, which is often valued by patients because it improves their functioning and allows them to engage in other treatments, albeit it also carries the risk of dependence. Medications do not improve coping with anxiety directly and they are effective only for as long as they are taken. Therefore, relapses following the cessation of pharmacotherapy are common and prevention of relapse is enhanced by the addition of treatments that teach patients strategies for effective and long-term coping with anxiety, for example, CBT. The key task in pharmacological treatment is to find the right balance between the effectiveness of the medication and its adverse effects.

The aim of pharmacotherapy of anxiety disorders is remission, which is defined as the minimal presence of symptoms, little or no anxiety-related distress and return to normal functioning. Remission may decrease the risk of relapse after medication discontinuation and is most likely achieved by using the medication in an adequate dose and long enough (usually for at least 6-12 weeks). If an adequate trial with the initial (first-line) medication produces no improvement, the patient can be administered another (second-line) pharmacological agent. If there is some improvement, an augmenting agent (usually the second- or third-line medication) can be added. Approximately 75% of patients respond to the initial low dose of antidepressants, but patients with anxiety disorders typically take longer to respond than those with depression (Bandelow et al., 2012).

Maintaining remission requires an ongoing, daily use of the medication for at least 6 months. If the remission has lasted for at least 6-12 months and the patient is ready for discontinuation of pharmacotherapy, medication taper can be planned. The duration of this gradual reduction in dose until the medication is ceased depends on the type of medication, presence of any anxiety or withdrawal symptoms and various personal circumstances, such as ability to tolerate distress and cope. As a rule, medication should not be ceased abruptly after long-term pharmacotherapy.

Selective serotonin reuptake inhibitors and serotonin and noradrenaline reuptake inhibitors are considered by the evidence-based treatment guidelines (Katzman et al., 2014) to be the first-line pharmacotherapy for panic disorder, social anxiety disorder and GAD. Second- or third-line medications include benzodiazepines, tricyclic antidepressants for panic disorder and GAD, classical, irreversible monoamine oxidase inhibitors for social anxiety disorder and pregabalin, agomelatine and buspirone for GAD. Antidepressants are not always effective, they do not work quickly and are often associated with adverse effects. For these reasons, benzodiazepines can be used as second- or third-line pharmacotherapy or as augmenting agents. They are generally effective at relieving the symptoms of anxiety and well tolerated, but need to be administered carefully because of their own adverse effects and dependence associated with long-term use and because their use does not always result in functional improvement.

Pharmacotherapy is of little value for specific phobia. Medication treatment of agoraphobia is useful to the extent that it is effective in treating the associated panic attacks. Pharmacotherapy appears to have a limited role for agoraphobia unrelated to panic attacks.

Cognitive Behaviour Therapy (CBT)

The main advantages of CBT over pharmacotherapy are that it enables patients to cope with anxiety effectively and that treatment gains are maintained post-treatment and even enhanced with the passage of time. It achieves this by helping patients change unhelpful beliefs about the dangers of anxiety and by assisting them to tolerate anxiety symptoms better and reduce avoidance of feared situations. Such changes improve patients' functioning and decrease their vulnerability to future anxiety and the risk of relapse after treatment cessation. In addition, CBT fosters an active attitude towards treatment, which allows patients to develop a sense of ownership of their treatment gains. For these reasons, therapeutic effects of CBT are more likely to last longer than those of pharmacotherapy.

The techniques of cognitive and behaviour therapy for anxiety disorders are usually combined in clinical practice. They can be administered in individual and group formats, and in recent times CBT has also been successfully delivered via the Internet.

The first step in CBT is psychoeducation. This includes provision of information about anxiety, correction of any misconceptions and identification of the factors that maintain the disorder, which serves as the basis for treatment. Cognitive therapy techniques target maladaptive, anxiety-related assumptions,

beliefs, misinterpretations and appraisals, which play a role in the development and maintenance of anxiety disorders. In panic disorder, these techniques are used to challenge and correct catastrophic misinterpretations of bodily symptoms and to modify beliefs about body-based threat and the dangerous nature of anxiety. In social anxiety disorder, cognitive therapy addresses the negative assumptions and beliefs about oneself, others and social situations, as well as perception of the social environment as threatening and judgemental. Cognitive therapy techniques for GAD include imaginal exposure to the content of worries, modification of beliefs about the benefit of worrying and exercises that aim to improve coping with uncertainty.

The goal of behaviour therapy is to change and eliminate behaviours (e.g., avoidance) that help maintain pathological anxiety and impair function. The key behaviour therapy technique is exposure to the situations or stimuli that elicit anxiety. It is particularly useful for phobic disorders because they are characterised by prominent avoidance. Habituation – a decrease in anxiety upon repeated exposure to phobic stimuli – is believed to be the key mechanism that accounts for the effectiveness of exposure therapy. Exposure is best tolerated when it is performed gradually so that patients face in a step-by-step fashion the phobic stimuli that elicit increasing levels of anxiety. Exposure should ideally be self-directed and *in vivo*, but depending on the nature of the phobia and patient characteristics, therapist-assisted and imaginal exposure may need to be conducted first. This may be combined with role-play and modelling of therapist's behaviour (e.g., holding an insect) during therapeutic sessions. Patients with panic disorder are instructed to induce the specific symptoms of autonomic hyperarousal and thus conduct exposure to these feared bodily stimuli; this technique is referred to as interoceptive exposure.

Symptom control techniques reduce bodily symptoms of anxiety and may be used in conjunction with CBT. They include various muscle relaxation techniques that can be useful for all anxiety disorders, especially GAD. Another symptom control technique is breathing retraining, which may be helpful in the treatment of panic patients with hyperventilation and other breathing difficulties. It is important that such techniques do not become "safety behaviours", used by patients for "prevention" of or distraction from anxiety. Research has shown that a focus on tolerating anxiety symptoms and being realistic about the consequences of anxiety is more effective than trying to prevent anxiety or panic. Social skills training, including assertiveness training, can be added to CBT in the treatment of social anxiety disorder when social skills may be lacking.

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