Anxiety disorders

Anxiety disorders are abnormal states in which the most striking features are mental and physical symptoms of anxiety, occurring in the absence of organic brain disease or another psychiatric .disorder

Symptoms of anxiety

Psychological arousal

Fearful anticipation, Irritability, Sensitivity to noise, Restlessness, Poor concentration, Worrying thoughts

Autonomic arousal

Gastrointestinal ● Dry mouth ● Difficulty in swallowing ● Epigastric discomfort ● Excessive wind ● Frequent or loose motions

Respiratory ● Constriction in the chest ● Difficulty inhaling

Cardiovascular ● Palpitations ● Discomfort in the chest ● Awareness of missed beats

Genitourinary ● Frequent or urgent micturition ● Failure of erection ● Menstrual discomfort

Muscle tension Tremor Headache Aching muscles

Hyperventilation Dizziness Tingling in the extremities Feeling of breathlessness

Sleep disturbance Insomnia Night terror

The symptom of anxiety is found in many disorders. In the anxiety disorders, it is the most severe and prominent symptom, and it is also prominent in the obsessional .disorders, although these are characterized by their striking obsessional symptoms

Although all of the symptoms can occur in any of the anxiety disorders, there is a characteristic pattern in each disorder, which will be described later. The disorders share many features of their clinical picture and aetiology, but there are also :differences

- In generalized anxiety disorders, anxiety is continuous, although it may fluctuate in intensity.
- In phobic anxiety disorders, anxiety is intermittent, arising in particular circumstances.
- In panic disorder, anxiety is intermittent, but its occurrence is unrelated to any particular circumstances.

Anxiety has long been recognized as a prominent symptom of many psychiatric disorders. Anxiety and depression often occur together and, until the last part of the nineteenth century, anxiety disorders were not classified separately from other mood disorders. It was Freud, in 1895, who first suggested that cases with mainly anxiety symptoms should be recognized as a separate entity under the name ‘anxiety .’neurosis

The classification of anxiety disorders

In ICD-10, anxiety disorders are divided into two named subgroups: (a) phobic ● anxiety disorder (F40); and (b) other anxiety disorder (F41), which includes panic .disorder and GAD

ICD-10 contains a category of mixed anxiety–depressive disorder, but DSM-5 does ● .not

Generalized anxiety disorder

Clinical picture

The symptoms of GAD are persistent and are not restricted to, or markedly increased in, any particular set of circumstances (in contrast to phobic anxiety disorders). All of the symptoms of anxiety can occur in GAD, but there is a characteristic pattern, which :consists of the following features

- Worry and apprehension that are more prolonged than in healthy people. The worries are widespread and are not focused on a specific issue as they are in panic disorder (i.e. on having a panic attack), social phobia (i.e. on being embarrassed), or OCD (i.e. on contamination). The person feels that these widespread worries are .difficult to control

- Psychological arousal, which may be manifested as irritability, poor concentration, and/or sensitivity to noise. Some patients complain of poor memory, but this is because of poor concentration. If true memory impairment is found, a careful search should be made for a cause other than anxiety.
- Autonomic overactivity, which is most often experienced as sweating, palpitations, dry mouth, epigastric discomfort, and dizziness.
- Muscle tension, which may be experienced as restlessness, trembling, inability to relax, headache (usually bilateral and frontal or occipital), and aching of the shoulders and back.
- Hyperventilation, which may lead to dizziness, tingling in the extremities and, paradoxically, a feeling of shortness of breath.

Sleep disturbances, which include difficulty in falling asleep and persistent worrying ● thoughts. Sleep is often intermittent, unrefreshing, and accompanied by unpleasant dreams. Some patients have night terrors and wake suddenly feeling extremely anxious. Early-morning waking is not a feature of GAD, and its presence strongly .suggests a depressive disorder

Other features, which include tiredness, depressive symptoms, obsessional ● symptoms, and depersonalization. These symptoms are never the most prominent feature of GAD. If they are prominent, another diagnosis should be considered

Diagnosis is usually made if a number of the above mentioned symptoms were found every day, most of the day for a duration that varies from several weeks to 6 months

Differential diagnosis

Depressive disorder

Anxiety is a common symptom in depressive disorder, and GAD often includes some depressive symptoms. The usual convention is that the diagnosis is decided on the basis of .the severity of two kinds of symptom and the order in which they appeared

Schizophrenia

People with schizophrenia sometimes complain of anxiety before the other symptoms are recognized. The chance of misdiagnosis can be reduced by asking anxious patients routinely .what they think caused their symptoms

Dementia

Substance misuse

Some people take drugs or alcohol to relieve anxiety. Patients who are dependent on drugs or alcohol sometimes believe that the symptoms of drug withdrawal are those of anxiety, and .take anxiolytic or other drugs to control them

Physical illness

Some physical illnesses have symptoms that can be mistaken for those of an anxiety disorder. This possibility should be considered in all cases, but especially when there is no obvious psychological cause of anxiety, or there is no history of past anxiety. The following :conditions are particularly important

In thyrotoxicosis, the patient may be irritable and restless, with tremor and tachycardia. ● Physical examination may reveal characteristic signs of thyrotoxicosis, such as an enlarged thyroid, atrial fibrillation, and exophthalmos. If there is any doubt, thyroid function tests should .be arranged

Phaeochromocytoma and hypoglycaemia usually cause episodic symptoms, and are ● therefore more likely to mimic a phobic disorder or panic disorder. However, they should also be considered as a differential diagnosis of GAD. If there is any doubt, appropriate physical .examination and laboratory tests should be carried out

Epidemiology

Estimates of incidence and prevalence vary according to the diagnostic criteria used in the survey, and whether a clinical significance criterion is used. The Adult Psychiatric Morbidity Survey found a 12-month prevalence of 4.4% for GAD in England. Rates in women are about twice as high as those in men. GAD is associated with several indices of social disadvantage, including lower household income and unemployment, as well as divorce and separation

Aetiology

Stressful events

Clinical observations indicate that GADs often begin in relation to stressful events, and .some become chronic when stressful problems persist

Early experiences

Accounts given by anxious patients of their experience in childhood suggest that early adverse experience is a cause of GAD. Parenting styles characterized by overprotection and lack of emotional warmth may also be a risk factor for GAD

.Psychoanalytic theories

Psychoanalytical theory proposes that anxiety arises from intrapsychic conflict when the ego :is overwhelmed by excitation from any of the following three sources

;the outside world (realistic anxiety) ●

;the instinctual levels of the id, including love, anger, and sex (neurotic anxiety) ●

.the superego (moral anxiety) ●

According to this theory, in GAD, anxiety is experienced directly unmodified by the defence mechanisms that are thought to be the basis of phobias or obsessions. The theory proposes that in GADs the ego is readily overwhelmed because it has been weakened by a development failure in childhood. Normally, children overcome this anxiety through secure relationships with loving parents. However, if they do not achieve this security, as adults they .will be vulnerable to anxiety when experiencing separation or potentially threatening events

Cognitive behavioural theories

Conditioning theories propose that GAD arises when there is an inherited predisposition to excessive responsiveness of the autonomic nervous system, together with generalization of the responses through conditioning of anxiety to .previously neutral stimuli

Cognitive theory. Particular coping and cognitive styles may also predispose individuals to the development of GAD, although it is not always easy to distinguish .predisposition from the abnormal cognitions that are seen in the illness itself

Personality

Neurobiological mechanisms

The neurobiological mechanisms involved in GAD are presumably those that mediate normal anxiety. The mechanisms are complex, involving several brain systems and several .neurotransmitters

Studies in animals have indicated a key role for the amygdala. The hippocampus is also believed to have an important role in the regulation of anxiety, because it relates fearful .memories to relevant present contexts

Noradrenergic neurons that originate in the locus coeruleus increase arousal and anxiety, whereas 5-HT neurons that arise in the raphe nuclei appear to have complex effects, and serve both to signal the presence of anxiety producing stimuli in the environment and also to restrain the associated behavioural responses. Gamma-aminobutyric acid (GABA) receptors, which are widely distributed in the brain, are inhibitory and reduce anxiety, as do the .associated benzodiazepine-binding sites

Genetic causes

Prognosis

One of the DSM-5 criteria for GAD is that the symptoms should have been present for 6 months. One of the reasons for this cut-off is that anxiety disorders that last for longer than 6 months have a poor prognosis. Thus most clinical studies suggest that GAD is typically a chronic condition with low rates .of remission over the short and medium term

Treatment

Self-help and psychoeducation

Relaxation training

Cognitive behaviour therapy

This treatment combines relaxation with cognitive procedures designed to help .patients to control worrying thoughts

Medication

Medication can be used to bring symptoms under control quickly while the effects of psychological treatment are awaited. It can also be used when psychological treatment has failed. However, medication is often prescribed too readily and for too .long

Short-term treatment. One of the longer-acting benzodiazepines, such as diazepam, is appropriate for the short-term treatment of GAD

Long-term treatment. Because GAD often requires lengthy treatment, for which benzodiazepines are unsuitable, and is often comorbid with depression and other anxiety disorders, treatment guidelines usually recommend selective serotonin reuptake inhibitors (SSRIs) as the initial choice

Phobic anxiety disorders

Phobic anxiety disorders have the same core symptoms as GAD, but these symptoms occur only in specific circumstances. In some phobic disorders these circumstances are few and the patient is free from anxiety for most of the time. In other phobic disorders many circumstances provoke anxiety, and consequently anxiety is more frequent, but even so there are some situations in which no anxiety is experienced. Two other features characterize phobic disorders. First, the person avoids circumstances that provoke anxiety and, secondly, they experience anticipatory anxiety when there is the prospect of encountering these circumstances. The circumstances that provoke anxiety can be grouped into situations (e.g. crowded places), ‘objects’ (a term that includes living things such as spiders), and natural phenomena (e.g. thunder). For clinical purposes, three principal phobic syndromes are .recognized—specific phobia, social phobia, and agoraphobia

Specific phobia

Clinical picture

A person with a specific phobia is inappropriately anxious in the presence of a particular object or situation. In the presence of that object or situation, the person experiences the symptoms of anxiety listed before. Anticipatory anxiety is common, and the person usually seeks to escape from and avoid the feared situation. Specific phobias can be characterized further by adding the name of the stimulus (e.g. spider phobia). In the past it was common to use terms such as arachnophobia (instead of spider phobia) or acrophobia (instead of phobia of heights), but this practice adds .nothing of value to the use of the simpler names

Specific phobia

In DSM-5, five general types of specific phobia are recognized, which are concerned :with

animals ●

aspects of the natural environment ●

blood, injection, medical care, and injury ●

.situations (for example, aeroplanes, lifts, enclosed spaces) ●

.other provoking agents (for example, fears of choking or vomiting) ●

Specific phobia

Epidemiology

Among adults, the lifetime prevalence of specific phobias has been estimated, using DSM-IV criteria, to be around 7% in men and 17% in women. The age of onset of most specific phobias is in childhood. The onset of phobias of animals occurs at an average age of 7 years, blood phobia at around 8 years, and most situational phobias .develop in the early twenties

Specific phobia

Aetiology

Persistence of childhood fears

Most specific phobias in adulthood are a continuation of childhood phobias. Specific phobias are common in childhood. By the early teenage years most of these childhood fears will have been lost, but a few persist into adult life. Why they persist is not .certain, except that the most severe phobias are likely to last the longest

Genetic factors

In one study, 31% of first-degree relatives of people with specific phobia also had the condition. Genetic vulnerability may involve differences in the strength of fear conditioning, which has a heritability of around 40%

Specific phobia

Psychoanalytical theories

These theories suggest that phobias are not related to the obvious external stimulus, but to an internal source of anxiety. The internal source is excluded from consciousness by repression and attached to the external object by displacement. This theory is not supported .by objective evidence

Conditioning and cognitive theories

Conditioning theory suggests that specific phobias arise through association learning. A minority of specific phobias appear to begin in this way in adulthood, in relation to a highly stressful experience. For example, a phobia of horses may begin after a dangerous encounter with a bolting horse. Some specific phobias may be acquired by observational learning, as the child observes another person's fear responses and learns to fear the same stimuli. Cognitive factors are also involved in the maintenance of the fear, especially fearful .anticipation of and selective attention to the phobic stimuli

Specific phobia

Prepared learning

This term refers to an innate predisposition to develop persistent fear responses to certain stimuli. Some young primates seem to be prepared to develop fears of snakes, but it is not certain whether the same process accounts for some of the specific .phobias of human children

Neural mechanisms

Functional imaging studies have revealed hyperactivity of the amygdala upon presentation of the feared stimulus, which appears to diminish with successful treatment. Anticipation of a phobic stimulus activates the anterior cingulate cortex and .the insular cortex

Specific phobia

Differential diagnosis

Diagnosis is seldom difficult. The possibility of an underlying depressive disorder should always be kept in mind, since some patients seek help for longstanding specific phobias when a depressive disorder makes them less able to tolerate their phobic symptoms. Obsessional disorders sometimes present with fear and avoidance .of certain objects (e.g. knives)

Prognosis

The prognosis of specific phobia in adulthood has not been studied systematically. Clinical experience suggests that specific phobias that originate in childhood continue for many years, whereas those that start after stressful events in adulthood have a .better prognosis

Specific phobia

Treatment

The main treatment is the exposure form of behaviour therapy. With this treatment, the phobia is usually reduced considerably in intensity and so is the social disability. However, it is unusual for the phobia to be lost altogether. The outcome depends importantly on repeated and prolonged exposure, and up to 25% of phobic patients decline exposurebased therapies. Some patients seek help soon before an important engagement that will be made difficult by the phobia. When this happens, a few doses of a benzodiazepine may be prescribed to relieve phobic anxiety until a treatment can .be arranged

Social phobia

Clinical picture

In this disorder, inappropriate anxiety is experienced in social situations, in which the person feels observed by others and could be criticized by them. Socially phobic people attempt to avoid such situations. If they cannot avoid them, they try not to engage in them fully—for example, they avoid making conversation, or they sit in the place where they are least conspicuous. Even the prospect of encountering the situation may cause considerable anxiety, which is often misconstrued as shyness. Social phobia can be distinguished from shyness by the levels of personal distress .and associated social and occupational impairment

Social phobia

The situations in which social phobia occurs include restaurants, canteens, dinner parties, seminars, board meetings, and other places where the person feels observed by other people. Some patients become anxious in a wide range of social situations (generalized social phobia), whereas others are anxious only in specific situations, such as public speaking, writing in front of others, or playing a musical instrument in public. In DSM-5 ‘performance only’ social phobia, anxiety is restricted to speaking or performing in public. (In DSM-5 the term ‘social anxiety disorder’ is preferred to ‘social phobia’.)

Social phobia

Differential diagnosis

Agoraphobia and panic disorder

Generalized anxiety disorder and depressive disorder

Schizophrenia

Body dysmorphic disorder

Avoidant personality disorder

Inadequate social skills

Normal shyness

Social phobia

Epidemiology

The National Comorbidity Survey Replication reported a lifetime prevalence rate of social phobia in the community of around 12%. The rate is therefore not much lower than that of specific phobia. Social phobias are about equally frequent among the men and women who seek treatment, but in community surveys they are reported rather more frequently by women

Aetiology

Genetic factors

Conditioning

Social phobia

Cognitive factors The principal cognitive factor in the aetiology of social phobia is an undue concern that other people will be critical of the person in social situations (often referred to as a fear of :negative evaluation). This concern is accompanied by several other ways of thinking, including

excessively high standards for social performance ●

negative beliefs about the self (e.g. ‘I’m boring’) ●

excessive monitoring of one’s own performance in social situations ● intrusive negative images of ● the self as supposedly seen by others. People with social phobia often adopt safety behaviours, such .as avoiding eye contact, which make it harder for them to interact normally

Neural mechanisms

Functional neuroimaging studies have found that patients with social phobia have increased .amygdala responses to presentation of faces with expressions of negative affect

Social phobia

Course and prognosis

Social phobia has an early onset, usually in childhood or adolescence, and can persist over many years, sometimes even into old age. Only about 50% of people with the disorder seek treatment, usually after many years of symptoms

Treatment

Psychological treatment

.Cognitive behaviour therapy is the psychological treatment of choice for social phobia

Drug treatment

Treatment guidelines generally recommend SSRIs as the first choice of pharmacological treatment in the management of social phobia. All of the SSRIs, as well as venlafaxine (an .SNRI), have been shown to be effective

Agoraphobia

Clinical features

Agoraphobic patients are anxious when they are away from home, in crowds, or in situations that .they cannot leave easily. They avoid these situations, feel anxious when anticipating them

The anxiety symptoms that are experienced by agoraphobic patients in the phobic situations are :similar to those of other anxiety disorders, although two features are particularly important

panic attacks, whether in response to environmental stimuli or arising spontaneously ●

.anxious cognitions about fainting and loss of control ●

The situations include buses and trains, shops and supermarkets, and places that cannot be left suddenly without attracting attention, such as the hairdresser's chair or a seat in the middle row of a theatre or cinema. As the condition progresses, the individual increasingly avoids these situations .until in severe cases they may be more or less confined to their home

Agoraphobia

Onset and course

The onset and course of agoraphobia differ in several ways from those of other phobic .disorders

Age of onset. In most cases the onset occurs in the early or mid-twenties, with a further period of high onset in the mid-thirties. In both cases this is later than the average ages of .onset of simple phobias (childhood) and social phobias (mostly the teenage years)

Circumstances of onset. Typically the first episode occurs while the person is waiting for public transport or shopping in a crowded store. Suddenly they become extremely anxious without knowing why, feel faint, and experience palpitations. They rush away from the place and go home or to hospital, where they recover rapidly. When they enter the same or similar .surroundings, they become anxious again and make another hurried escape

Agoraphobia

Differential diagnosis

Social phobia. Generalized anxiety disorder. Panic disorder. Depressive disorder. Paranoid disorders

Epidemiology

A recent community investigation in Europe, using strict DSM-IV criteria, estimated a .lifetime prevalence of agoraphobia without panic of 0.6%

Agoraphobia

Aetiology

Theories of the aetiology of agoraphobia have to explain both the initial anxiety attack .and its spread and recurrence. These two problems will now be considered in turn

Theories of onset: Agoraphobia begins with anxiety in a public place—generally, but .not always, as a panic attack. There are two explanations for the initial anxiety

- The cognitive hypothesis proposes that the anxiety attack develops because the person is unreasonably afraid of some aspect of the situation or of certain physical symptoms that are experienced in the situation
- The biological theory proposes that the initial anxiety attack results from chance environmental stimuli acting on an individual who is constitutionally predisposed to .over-respond with anxiety

Agoraphobia

Theories of spread and maintenance

Learning theories. Conditioning could account for the association of anxiety with increasing numbers of situations, and avoidance learning could account for the .subsequent avoidance of these situations

Personality. Agoraphobic patients are often described as dependent, and prone to avoiding rather than confronting problems. This dependency could have arisen from overprotection in childhood, which is reported more often by agoraphobic individuals .than by controls

Agoraphobia

Prognosis

Although short-lived cases may be seen in general practice, agoraphobia that has lasted for 1 .year generally remains for the next 5 years, and usually the illness runs a chronic course

Treatment

Much of the available treatment has been developed for panic disorder and for panic disorder with agoraphobia probably because, patients with agoraphobia without panic are not .common in clinical samples

Psychological treatment

Exposure treatment was the first of the behavioural treatments for agoraphobia. It was shown to be effective, but more so when combined with anxiety management

Cognitive behaviour therapy

Agoraphobia

Medications

Anxiolytic drugs. Benzodiazepines may be used for a specific, short-term purpose such as helping a patient to undertake an important engagement before other .treatment has taken effect

Antidepressant drugs. As well as the obvious use to treat a concurrent depressive disorder, antidepressant drugs have a therapeutic effect in agoraphobic patients who are not depressed but who have frequent panic attacks. Imipramine was one of the first agents to be used in this way, but similar effects have been reported with clomipramine, several SSRIs and venlafaxine have been shown to be effective in .panic disorder with and without agoraphobia

Panic disorder

Although the diagnosis of panic disorder did not appear in the nomenclature until 1980, similar cases have been described under a variety of names for more than a century. The central feature is the occurrence of panic attacks. These are sudden attacks of anxiety in which physical symptoms predominate, and they are .accompanied by fear of a serious medical consequence such as a heart attack

In the past, these symptoms have been variously referred to as irritable heart, Da Costa’s syndrome, neurocirculatory asthenia, disorderly action of the heart, and effort .syndrome

These early terms assumed that patients were correct in fearing a disorder of cardiac function. Some later authors suggested psychological causes, but it was not until the Second World War (when interest in the condition revived) that the cardiologist, Paul Wood (1907–1962) showed convincingly that the condition was a form of anxiety disorder. From then until 1980, patients with panic attacks were classified as having .either generalized or phobic anxiety disorders

In DSM-5 agoraphobia and panic disorder are diagnosed separately and patients who have both conditions receive both diagnoses. Panic disorder is included in ICD-10, but when patients have concomitant agoraphobia they are diagnosed as suffering from .agoraphobia with panic disorder

Clinical features

Symptoms of a panic attack

:Sudden onset of

Palpitations, Choking sensations, Chest pain, Dizziness and faintness

Depersonalization, Derealization

Fear of dying, losing control, or going mad

Not every patient has all of these symptoms during the panic attack and, for a diagnosis of panic disorder, DSM-5 requires the presence of only four or more .symptoms

:The important features of panic attacks are that

anxiety builds up quickly ●

the symptoms are severe ●

.the person fears a catastrophic outcome ●

Some people with panic disorder hyperventilate, and this adds to their symptoms. Hyperventilation is breathing in a rapid and shallow way that leads to a fall in the .concentration of carbon dioxide in the blood

:The resulting hypocapnia may cause the following symptoms

Dizziness, Tinnitus, Headache, Feeling of weakness, Faintness, Numbness

Tingling in the hands, feet, and face

Carpopedal spasms, Precordial discomfort, Feeling of breathlessness

The last symptom in the list, the feeling of breathlessness, is paradoxical, as the person is breathing excessively. It is important because it leads to a further increase in breathing, which worsens the condition. Hyperventilation should always be borne in mind as a cause of unexplained bodily symptoms. The diagnosis can usually be made .by watching the pattern of breathing when the symptoms are present

Diagnostic criteria

:In DSM-5 the diagnosis of panic disorder is made when

panic attacks occur recurrently (at least twice) and unexpectedly (i.e. not in response to an identified phobic stimulus); and

at least one attack has been followed by 4 weeks or more of persistent fear of (2) another attack and worry about its implications (e.g. having a heart attack), and/or a significant maladaptive change in behaviour (for example, avoiding exercise or public .transport)

Differential diagnosis

Panic attacks occur in GADs, phobic anxiety disorders (most often agoraphobia), depressive disorders, and acute organic disorder. Two of the DSM-5 diagnostic .criteria help to distinguish these secondary attacks from panic disorder

Epidemiology

The National Comorbidity Survey Replication found a 12-month prevalence rate of .DSM-IV panic disorder of 2.7% and a lifetime risk of 4.7%

Aetiology

Genetics

Panic disorder is familial, with about a fivefold increase in risk in first degree relatives. Rates in monozygotic twins are higher than those in dizygotic twins, indicating that the family aggregation is likely to be at least partly owing to genetic factors, with a .heritability of about 40%

Biochemistry

Chemical agents, notably sodium lactate and the noradrenaline α2 - adrenoceptor antagonist, yohimbine, but also flumazenil (a benzodiazepinereceptor antagonist), and a 5-HT receptor agonist, m-chlorophenylpiperazine, can induce panic attacks more readily in patients with panic disorder than in healthy individuals. The multitude of chemical agents that provoke panic attacks in panic disorder patients make it difficult to identify a single causal mechanism, and the abnormal responses could reflect altered psychological attribution, as described below. However, changes in 5-HT, noradrenaline, and GABA-ergic mechanisms have been suggested. There is most evidence for changes in GABA, with lowered cortical GABA levels measured by magnetic resonance spectroscopy, as well as diminished benzodiazepine-receptor .binding in the parietotemporal regions in unmedicated patients with panic disorder

Neural mechanisms

Animal experimental studies suggest that the neural circuitry of fear and particularly escape behaviour (which panic attacks resemble to some extent) involves the amygdala, periaqueductal grey, hippocampus, hypothalamus, and brainstem nuclei, .including the locus coeruleus, the origin of noradrenaline cell bodies

Cognitive theories

The cognitive hypothesis is based on the observation that fears about serious physical or mental illness are more frequent among patients who experience panic attacks than among anxious patients who do not have panic attacks. It is proposed that there is a spiral of anxiety in panic disorder, as the physical symptoms of anxiety activate fears of illness and thereby generate more anxiety. ‘Safety behaviours’ prevent .disconfirmation of these fears

Hyperventilation as a cause

A subsidiary hypothesis proposes that hyperventilation is a cause of panic disorder. Although there is no doubt that voluntary overbreathing can produce a panic attack, it .has not been shown that panic disorder is caused by involuntary hyperventilation

Course and prognosis

Follow-up studies have generally included patients with panic attacks and .agoraphobia as well as patients with panic disorder alone

More recent studies of patients diagnosed with panic disorder also reveal a lengthy course, with fluctuating anxiety and depression. About 30% of patients remit without subsequent relapse, and a similar proportion show useful improvement, although with persistent symptomatology

Treatment

Apart from supportive measures and help with any causative life problems, treatment is with medication or cognitive therapy. A number of different kinds of medication can .be used

Benzodiazepines

When given in high doses, benzodiazepines control panic attacks. In these doses, most benzodiazepines cause sedation, but alprazolam, a high-potency compound, is an exception. For this reason it has been used to treat panic disorder, and its effectiveness over placebo has been demonstrated in many controlled trials. At the end of treatment, alprazolam should be reduced very gradually to avoid withdrawal symptoms, because even when it is reduced over 30 days, about one-third of patients .report significant withdrawal symptoms

Antidepressants

Imipramine was the first antidepressant to be shown to be effective for the treatment of panic disorder, and other tricyclic antidepressants, including clomipramine and lofepramine, are superior to placebo in controlled trials. SSRIs are also beneficial in the treatment of panic disorder, as is the SNRI, venlafaxine, and the selective noradrenaline re-uptake inhibitor, reboxetine. The initial effect of antidepressants in patients with panic disorder is often to produce an unpleasant feeling of apprehension, .sleeplessness, and palpitations. For this reason the initial dose should be small

Cognitive therapy

Cognitive therapy reduces the fears of the physical effects of anxiety, which are thought to provoke and maintain the panic attacks. Common fears of this kind are that palpitations indicate an impending heart attack, or that dizziness indicates impending loss of consciousness. In treatment, the physical symptoms that the patient fears are induced by hyperventilation or exercise. The therapist points out the sequence of physical symptoms that leads to fear, and explains that a similar sequence occurs in the early stages of a panic attack. The therapist goes on to question the patient's .belief in the feared outcome