Anxiety and Anxiety Medication: A Clinician Guide

Dr Adam Lake, GP | Last updated June 2026

Introduction

This guide is the referenced companion to two patient-facing leaflets — one on understanding anxiety, one on medication for anxiety. It sets out the evidence and reasoning behind those leaflets, so that the claims made to patients can be traced to source and discussed with colleagues. It is intended to support clinician understanding and shared decision-making, not to replace national guidance.

I have written it from my reading of the literature on the physiology of anxiety, the diagnostic framework, and the evidence for the main drug classes used in anxiety — alongside my experience as a GP with an interest in mental health. Where the evidence is weak, contested or absent, I have tried to say so plainly rather than imply more certainty than exists.

Part One: Understanding Anxiety

Anxiety as an adaptive threat response

Anxiety is not a disease with a single identifiable biological lesion in the way an infection or tumour is. It is better understood as a state produced by the body's threat-and-safety system — the coordinated set of neural and endocrine responses that evolved to detect danger and prepare the organism for action.[1,2] When the brain appraises a possible threat, whether physical, emotional or social, it triggers changes throughout the body: increased heart rate and respiration, raised muscle tension, heightened vigilance, diverted digestion, and a narrowing of attention toward the perceived danger.[2]

In genuine danger this response is protective. Problems arise when it activates too readily, too strongly, or for too long — particularly when no physical action resolves the threat. A useful framing is of an alarm system that has become over-sensitive: functioning as designed, but calibrated to trigger on cues that do not warrant it.[1] Framing anxiety as something the nervous system does, rather than a fixed entity a person has, fits the evidence better and tends to reduce secondary fear of the symptoms themselves.

Physical symptoms and the symptom-fear cycle

Because the threat response is primarily somatic, anxiety is largely experienced in the body: palpitations, breathlessness or air hunger, dizziness, trembling, sweating, nausea, and sensations of unreality or detachment. These are downstream effects of adrenergic activation and altered respiration — for example, the tingling and light-headedness of hyperventilation reflect a fall in blood carbon dioxide rather than any lack of oxygen.[2] One of the most clinically important features of anxiety is that these sensations can themselves

be appraised as threatening, which further activates the stress response and intensifies symptoms in a self-reinforcing cycle. Recognising and explaining this cycle is often the first therapeutic step.[4]

Effects on thinking and attention

A threat state also shifts cognition. Rapid threat-detection circuitry becomes more dominant, while systems for reflection, planning and perspective-taking become relatively less so.[1,3] Clinically this presents as worry, catastrophic prediction, difficulty concentrating, racing or intrusive thoughts, and heightened self-monitoring. Worry can be understood as the mind attempting to pre-empt danger; it becomes exhausting because the system is sustained for far longer than the brief activation it evolved for.[3] Intrusive thoughts are distressing precisely because they are unwanted, and their presence does not signal intent. As arousal settles, thinking usually becomes more flexible again.

Why anxiety persists

Anxiety has evolved to be a helpful self-limiting response to threatening situations, yet several well-described processes can keep the system activated beyond its useful course, and come to cause severe distress and impact day to day functioning. Repeated activation can sensitise the threat system, so that progressively smaller cues — including internal sensations — trigger a response.[1,2] Increased monitoring makes ordinary sensations feel significant. Avoidance brings short-term relief but denies the nervous system the chance to relearn safety, so the range of situations that feel threatening tends to expand over time.[4] Fear of the sensations themselves can sustain anxiety even when external circumstances improve. Sleep disruption, illness, pain, caffeine, hormonal change and chronic stress all lower the threshold for activation.

Stress, life circumstances and trauma

The nervous system is highly responsive to life context. In modern life most threats are psychological and social rather than physical, but the body responds to them with the same machinery, producing prolonged activation.[2] People tend to function best with a reasonable sense of safety, predictable rhythm, supportive relationships, meaningful activity, opportunity for recovery, and some sense of control; sustained strain across several of these can hold the system in a heightened state. Anxiety frequently develops gradually as demands outpace recovery, without any single precipitating event.

For some people anxiety is linked to earlier overwhelming experience. During high stress the nervous system learns rapidly to protect against future harm, which can leave it more reactive to cues resembling the original experience.[1] Not everyone with anxiety has identifiable trauma, and anxiety arises for many reasons; but where past experience is relevant, this learning frame helps explain why anxiety can feel automatic. With safety, supportive relationships and appropriate therapy, the system can relearn that present situations differ from past threats.

Diagnosis and what it means

Diagnostic terms such as generalised anxiety disorder, panic disorder and social anxiety disorder are useful for communication, for accessing treatment and adjustments, and for validation.[5] But unlike conditions confirmed by a test, these categories are derived by expert consensus from observed clusters of symptoms; there is no biological marker that confirms a person "has anxiety."[4,5] They are descriptive rather than explanatory. People with the same label may have arrived there by very different routes, so the individual context matters as much as the category. A diagnosis is a shared language, not an account of cause, and receiving one does not imply a permanent defect.

As all clinicians of course know, it is worth considering the physical conditions and substances that can mimic or drive anxiety symptoms — thyroid disease, arrhythmias and other cardiac causes, excess caffeine, alcohol and stimulant use or withdrawal, and medications such as salbutamol, steroids and some decongestants. A focused history, examination and, where indicated, basic investigations (including thyroid function) help avoid attributing a treatable physical cause to anxiety.[6]

Supporting recovery

Recovery usually reflects the nervous system spending more time in states of safety and regulation, and tends to come from consistent influences rather than a single intervention.[4] Understanding the mechanism reduces secondary fear. Predictable routines, regular movement, and more settled breathing patterns all support regulation. Psychological therapies — cognitive behavioural approaches in particular have the strongest evidence base — help reduce avoidance and rebuild confidence in tolerating symptoms.[4,6] Supportive relationships matter, since the nervous system responds to signals of safety from others. Medication can reduce symptoms for some people and is discussed in detail below. Recovery is rarely linear; fluctuation with stress, illness or fatigue is expected. For many, the aim is a changed relationship with anxiety — its return to a useful signal rather than a constant alarm — rather than the complete absence of anxious feeling.

Psychological therapy and how it compares with medication

Because this is partly a guide to medication, it is worth stating plainly where drugs sit relative to psychological therapy. Across the main anxiety disorders, pharmacological and psychological treatments have broadly comparable average effect sizes, and combined treatment is often, though not always, superior to either alone.[9] Cognitive behavioural therapy has the strongest and most consistent evidence base, but it is not the only effective approach.[4,6] For a GP the practical point is that medication is not the only first-line option and need not be the default; NICE supports a stepped approach in which low-intensity psychological interventions, guided self-help and CBT are offered alongside or ahead of medication, according to severity and patient preference.[6]

The common belief that therapy's benefits endure while medication's are lost on stopping is less clear-cut than often assumed. A meta-analysis of follow-up studies found that gains from CBT and other psychotherapies were largely maintained for up to two years — but also that medication, and to a lesser extent placebo, showed enduring effects, and that the difference between them was not significant.[18] That finding has been contested on methodological grounds, so it shouldn't be over-read; the fair summary is that durable benefit is possible from both. Relapse (or withdrawal in some cases after stopping antidepressants) after stopping either is common.

Part Two: Medication for Anxiety

Medication is one option among several and is rarely the whole answer. Trial data describe averages across populations: within them, some people benefit substantially, some little or not at all, and some are harmed or feel worse. Group averages cannot predict the individual response, though they help set realistic expectations.

A practical point before the drug classes: anxiety and depression co-occur very commonly, and mixed anxiety-depression is one of the most frequent presentations in primary care. The SSRIs and SNRIs are first-line for both, which often simplifies the choice where the two coexist; the antidepressant guides on this site cover their use, mechanism and adverse effects in more depth, and apply directly here. Where anxiety is the predominant problem, the considerations in this guide may be useful; where depression is predominant or there is significant risk, approaches are covered in other documents.

Antidepressants (SSRIs and SNRIs)

SSRIs and SNRIs are the usual first-line pharmacological treatment for longer-term anxiety disorders.[6,8] As with their use in depression, they do not correct a known chemical imbalance; they alter monoaminergic signalling, and appear over weeks to change how strongly the brain and nervous system respond to threat and emotional signals.[7] A reasonable way to put this to patients is that they may turn down the volume of anxiety rather than remove it, and may make other forms of support more accessible, without resolving the reasons anxiety developed. There are separate antidepressant leaflets on this site; the mechanism, myths and adverse effects covered there apply here too.

Across randomised trials, SSRIs and SNRIs outperform placebo for anxiety disorders, but the average benefit is modest and broadly comparable to that seen for antidepressants in depression. Cross-condition comparison is imprecise — the disorders are rated on different scales and differ in placebo response — and the sometimes-quoted claim that anxiety responds more strongly than depression rests largely on uncontrolled pre-post effect sizes rather than placebo-controlled differences.[9] In generalised anxiety disorder specifically, network meta-analysis confirms benefit over placebo for several agents, with duloxetine, venlafaxine and escitalopram among those with supporting evidence; expressed as number needed to treat, roughly one extra person benefits for every five to seven treated, though

estimates vary by disorder, drug, and how response is defined. In generalised anxiety disorder specifically, network meta-analysis confirms benefit over placebo for several agents — duloxetine, pregabalin, venlafaxine and escitalopram have the best-supported evidence — though the placebo-controlled difference is small, of the order of two to three points on the 56-point Hamilton anxiety scale.[10] Translated into a number needed to treat, this is commonly approximated as one extra person benefiting for every five to seven treated, though the figure varies with disorder, drug and outcome definition and is best read as a rough guide. These figures should be read with the same caution as in depression: much of the apparent variation between drugs reflects analytic choices, and several reviewers have noted high risk of bias across the underlying trials.[10,11]

What patients notice varies. Some report reduced intensity or frequency of anxiety, fewer panic symptoms, better sleep and greater emotional steadiness; others describe emotional blunting or feeling flattened; some notice no meaningful benefit.[15]

A temporary increase in anxiety in the first one to two weeks is common, particularly with SSRIs, and can be distressing if not anticipated; it settles in most but not all people.[6,8] Common adverse effects include nausea, headache, sleep disturbance, early restlessness and sexual dysfunction. Some settle with time; others persist. Sexual dysfunction in particular is common and under-reported, and is covered more fully in the antidepressant guide.

SSRIs and SNRIs can cause withdrawal symptoms on stopping or dose reduction, including dizziness, anxiety or agitation, flu-like symptoms, sleep disturbance and sensory symptoms such as “electric shock” sensations.[12,13] These are frequently mistaken for a return of anxiety. Gradual, individualised tapering reduces the risk and should be planned in advance; the hyperbolic-tapering principle discussed in the antidepressant guide applies equally here.[12]

Pregabalin and gabapentinoids

Pregabalin is licensed for generalised anxiety disorder and shows efficacy over placebo in trials, but in practice its risks have come to outweigh that role for most prescribers. Since April 2019 it has been a Class C controlled drug (Schedule 3) in the UK, reflecting dependence, misuse and diversion, and it now carries one of the fastest-rising drug-death tolls in the country.[6,14] Current guidance positions it as a later-line option only, to be considered after SSRIs and SNRIs and with careful screening for any history of substance misuse beforehand; abrupt cessation can precipitate rebound anxiety and seizures. It is not initiated in primary care for anxiety, and I would regard any decision to do so as one for specialist input rather than routine primary care. It is included here for completeness rather than as a recommendation.

Benzodiazepines

Benzodiazepines are no longer recommended for the routine treatment of anxiety disorders and are prescribed only rarely, in exceptional short-term situations.[6] They rapidly reduce anxiety in the short term, but the benefit does not persist after stopping, tolerance develops, and dependence is common with risk rising markedly as use continues; withdrawal can be difficult and protracted.[14] Crucially, they do not help the nervous system relearn safety over time, and may impede it. Where used at all, this should be a carefully limited, time-bounded prescription with an explicit review plan.[6,14]

Buspirone

Buspirone is a 5-HT1A partial agonist licensed for generalised anxiety disorder. It is non-sedating, does not cause physical dependence or a withdrawal syndrome, and has a benign safety profile. Its drawbacks are a delayed onset of two to four weeks, an evidence base limited by small and dated trials, and little role outside GAD.[6,15] In practice it is not a routine primary-care medication: on most UK formularies it is restricted to specialist (mental health team) initiation, with any continuation in primary care under shared-care arrangements. It is included here for completeness only rather than as a primary-care option.

Antihistamines

Some sedating antihistamines, principally hydroxyzine, are sometimes used for short-term or situational anxiety. There is some evidence that hydroxyzine is more effective than placebo in generalised anxiety disorder, but the Cochrane review judged the trials at high risk of bias, few in number and small, and concluded it cannot be recommended as a reliable first-line treatment.[16] They are best understood as providing short-term calming or sedation rather than modifying the underlying course of anxiety. Onset is usually within about 30 minutes with effects lasting several hours; sedation, dry mouth and next-day grogginess are the main limitations, with implications for driving.

Beta-blockers

Beta-blockers such as propranolol reduce the peripheral, adrenaline-driven physical symptoms of anxiety — palpitations, tremor, sweating — and act on the body rather than on thought or worry directly. They are often used situationally, for example for performance anxiety, and need not be taken regularly.[6] It is worth being honest about the evidence base, however: the most thorough systematic review and meta-analysis concluded that the quality of evidence is insufficient to support the routine use of propranolol in any anxiety disorder, with most supporting data old and small.[17] The rationale that reducing bodily symptoms can interrupt the symptom-fear cycle is plausible and consistent with the mechanism described in Part One, but it should not be presented to patients as well-established efficacy. Beta-blockers are contraindicated in asthma and some other conditions.

Particular populations

A few groups warrant extra caution. In older adults, SSRIs carry a higher risk of hyponatraemia and falls, sedating antihistamines add anticholinergic burden, and benzodiazepines and Z-drugs are best avoided. Starting doses are often lower.[6] In children and young people, the increased risk of suicidal thoughts and behaviour early in antidepressant treatment — well described for depression — applies here too, so initiation and monitoring usually sit with specialist services. In the perinatal period, decisions involve balancing the risks of untreated anxiety against medication exposure, and where required perinatal mental health services are available for advice or referrals.

Stepped care and when to refer

Anxiety is mostly managed in primary care, within a stepped-care framework: psychoeducation and active monitoring for milder presentations; low-intensity psychological interventions and guided self-help next; then high-intensity therapy (typically CBT) and/or medication for moderate to severe or persistent symptoms.[6] In England, NHS Talking Therapies (formerly IAPT) can be accessed by self-referral as well as GP referral, which is often the quickest route to evidence-based therapy.

Specialist referral is worth considering where there is diagnostic uncertainty, marked functional impairment or risk, inadequate response to two adequate treatment trials, significant comorbidity (including substance use), or where a treatment is contemplated that sits outside routine primary care. Urgent assessment is warranted where anxiety is accompanied by suicidal thoughts, inability to maintain basic safety, or a level of distress the person cannot manage.

Putting medication in context

Medication can reduce symptoms enough to improve sleep, lessen panic, and make other forms of support more accessible. What it does not do is address why the nervous system is activated, the context in which anxiety developed, or the patterns that maintain it. For many people it is most useful alongside psychological and social approaches rather than instead of them.[4,6]

There is no single right decision about medication for anxiety. Good decision-making means being clear about what the evidence shows and its limits, discussing adverse effects and withdrawal honestly, reviewing regularly, and revisiting the decision if something is not helping or no longer needed. As with antidepressants in depression, framing a medication trial as exactly that — a time-limited trial within an ongoing consent process — fits the evidence better than presenting it as a predictable or corrective treatment.

References

1. LeDoux JE. Rethinking the emotional brain. *Neuron.* 2012;73(4):653–676.
2. McEwen BS. Physiology and neurobiology of stress and adaptation: central role of the brain. *Physiological Reviews.* 2007;87(3):873–904.

3. Thayer JF, Lane RD. A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders.* 2000;61(3):201–216.
4. Craske MG, Stein MB. Anxiety. *The Lancet.* 2016;388(10063):3048–3059.
5. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (5th ed.).* American Psychiatric Publishing; 2013.
6. National Institute for Health and Care Excellence. *Generalised anxiety disorder and panic disorder in adults: management.* Clinical guideline CG113. 2011 (updated 2020).
7. Moncrieff J, Cohen D. Rethinking models of psychotropic drug action. *Psychotherapy and Psychosomatics.* 2005;74(3):145–153.
8. Baldwin DS, Anderson IM, Nutt DJ, et al. Evidence-based pharmacological treatment of anxiety disorders, post-traumatic stress disorder and obsessive-compulsive disorder: a revision of the 2005 guidelines from the British Association for Psychopharmacology. *Journal of Psychopharmacology.* 2014;28(5):403–439.
9. Bandelow B, Reitt M, Röver C, Michaelis S, Görlich Y, Wedekind D. Efficacy of treatments for anxiety disorders: a meta-analysis. *International Clinical Psychopharmacology.* 2015;30(4):183–192.
10. Slee A, Nazareth I, Bondaronek P, Liu Y, Cheng Z, Freemantle N. Pharmacological treatments for generalised anxiety disorder: a systematic review and network meta-analysis. *The Lancet.* 2019;393(10173):768–777.
11. Cipriani A, Furukawa TA, Salanti G, et al. Comparative efficacy and acceptability of 21 antidepressant drugs for the acute treatment of adults with major depressive disorder: a systematic review and network meta-analysis. *The Lancet.* 2018;391(10128):1357–1366.
12. Horowitz MA, Taylor D. Tapering of SSRI treatment to mitigate withdrawal symptoms. *The Lancet Psychiatry.* 2019;6(6):538–546.
13. Davies J, Read J. A systematic review into the incidence, severity and duration of antidepressant withdrawal effects. *Addictive Behaviors.* 2019;97:111–121.
14. Lader M. Benzodiazepines revisited — will we ever learn? *Addiction.* 2011;106(12):2086–2109.
15. Bandelow B, Michaelis S, Wedekind D. Treatment of anxiety disorders. *Dialogues in Clinical Neuroscience.* 2017;19(2):93–107.
16. Guaiana G, Barbui C, Cipriani A. Hydroxyzine for generalised anxiety disorder. *Cochrane Database of Systematic Reviews.* 2022;(3):CD006815.
17. Steenen SA, van Wijk AJ, van der Heijden GJMG, van Westrhenen R, de Lange J, de Jongh A. Propranolol for the treatment of anxiety disorders: systematic review and meta-analysis. *Journal of Psychopharmacology.* 2016;30(2):128–139.
18. Bandelow B, Sagebiel A, Belz M, Görlich Y, Michaelis S, Wedekind D. Enduring effects of psychological treatments for anxiety disorders: meta-analysis of follow-up studies. *The British Journal of Psychiatry.* 2018;212(6):333–338.