

# Adult ADHD

A lifelong brain-wiring difference where the prefrontal cortex (the control center at the front of your brain) cannot hold enough dopamine (the brain messenger that helps you lock onto what matters and finish what you start) or norepinephrine (the brain messenger that keeps you alert and ready to act) in the tiny gaps between nerve cells, so paying attention, stopping impulses, planning tasks, remembering instructions, and sitting still all fail—at work, at home, and everywhere in between.

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## THE STORY IN ONE SENTENCE

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“Adult ADHD means your brain's control tower runs low on dopamine and norepinephrine, so the attention spotlight, the impulse brake, and the mental scratch pad all break down at once.”

## Normal anatomy — the body's job

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Normally, the prefrontal cortex (the brain's executive control center at the front of the skull) uses dopamine and norepinephrine to filter out unimportant sights and sounds, hold information in working memory (your mental scratch pad for seconds to minutes), delay impulses until you decide whether to act, put tasks in the right order, and sense how much time is passing.

**BODY'S JOB**

The prefrontal cortex is the brain's CEO: it decides what to pay attention to, when to act, what to ignore, what comes next, and when to pump the brakes—so you can work toward goals step by step without getting sidetracked, acting on every impulse, or losing track of time.

1. A stimulus—a sound, a thought, something you see, or a memory—arrives at the prefrontal cortex.
2. Dopamine tags the important signals and suppresses the unimportant ones.
3. Norepinephrine keeps the prefrontal cortex alert, awake, and ready to respond.
4. Working memory holds information for seconds to minutes while you use it.
5. Impulse-control circuits delay action until the prefrontal cortex checks the consequences.
6. The internal clock and sequencing system track time and put tasks in order.
7. The result: you focus on one task, ignore distractions, finish what you start, stop yourself from acting too quickly, remember what you were told, sense time passing, and stay calm when things go wrong.

**What breaks**

In ADHD, the prefrontal cortex cannot hold or use dopamine and norepinephrine well enough, so the attention filter, the impulse brake, working memory, time sense, and emotional control all fail at the same time—in every part of life.

**CLINICAL TERM**

Dysregulation of dopamine and norepinephrine reuptake and receptor signaling in the prefrontal cortex, basal ganglia (the deep brain structures that start and stop actions), and cerebellum (the back-of-the-brain center that times movements and thoughts)—driven by genetic variants in DAT1 (the dopamine transporter gene), DRD4 and DRD5 (dopamine receptor genes), NET (the norepinephrine transporter gene), and COMT (the enzyme that breaks down dopamine)—impairs executive functions including sustained attention, response inhibition, working memory, time perception, and emotional regulation, resulting in inattention, impulsivity, hyperactivity, and functional impairment across multiple domains (work, home, relationships) that cannot be better explained by another psychiatric, neurological, or medical condition.

## How the body compensates

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Before diagnosis, many adults build routines—like drinking extreme amounts of coffee, hyperfocusing on high-interest tasks, relying on partners or apps to remember everything, or waiting until panic forces focus—that hide the problem until stress, new demands, or loss of external support overwhelm those workarounds and everything falls apart.

1. {'strategy': 'Self-medicating with caffeine, energy drinks, nicotine, or even illicit stimulants like cocaine or borrowed ADHD pills to boost focus temporarily.', 'why': 'These substances raise dopamine, norepinephrine, or both just enough to mimic normal attention and alertness for a short time—but the effect is unpredictable, wears off fast, requires higher and higher doses, and can lead to dependence, high blood pressure, insomnia, anxiety, or addiction.'}
2. {'strategy': 'Choosing high-stimulation jobs or environments that provide constant novelty, variety, urgency, or physical activity.', 'why': 'Novelty and urgency spike dopamine naturally, so emergency rooms, sales, creative work, journalism, startup companies, or hands-on trades keep attention engaged and hide the deficit—until the job becomes routine, boring, or requires sustained paperwork and planning, and focus collapses again.'}
3. {'strategy': 'Relying on partners, coworkers, parents, apps, alarms, sticky notes, or written lists to remember tasks, deadlines, appointments, and instructions.', 'why': 'External memory and reminders substitute for broken working memory—so the person seems to function fine until that support is removed (the partner leaves, the phone dies, the helpful coworker quits, or the job demands independent follow-through and self-direction).'}}
4. {'strategy': 'Procrastinating until a deadline creates panic and adrenaline, then hyperfocusing under extreme pressure at the very last minute.', 'why': 'Adrenaline (the body's main fight-or-flight hormone) and the stress hormone cortisol raise norepinephrine enough to temporarily restore focus and override distractions—so the person learns that panic is the only reliable way to get things done—but this pattern causes chronic stress, burnout, mistakes, all-nighters, and repeated crises that damage health, relationships, and reputation.'}
5. {'strategy': 'Avoiding tasks that require sustained attention, like paperwork, long meetings, detailed planning, reading dense material, balancing checkbooks, or organizing spaces.', 'why': 'The person builds a life around natural strengths—talking, improvising, troubleshooting, hands-on work, teaching—and sidesteps weaknesses, so ADHD stays hidden until a promotion, graduate school, parenting toddlers, managing a team, or a new relationship demands exactly what ADHD makes hardest.'}

6. {'strategy': 'Using constant movement, fidgeting, music, background noise, pacing, or standing desks to stay alert and awake during boring or long tasks.', 'why': 'Physical activity and sensory stimulation raise norepinephrine and dopamine just enough to keep the brain awake and the prefrontal cortex online—so tapping feet, clicking pens, chewing gum, pacing while on the phone, or playing music becomes a necessary coping tool to prevent the brain from shutting down, not just a habit or preference.'}
7. {'strategy': 'Hyperfocusing intensely on high-interest activities—video games, hobbies, research rabbit holes, creative projects—for hours without breaks, food, or sleep.', 'why': 'High-interest tasks flood the brain with dopamine, so attention locks on and will not let go—this can look like great focus, but it is actually uncontrolled focus that ignores time, body signals, and responsibilities, and leaves no energy for less interesting but equally important tasks like paying bills, eating meals, or sleeping on time.'}

## Domino effect

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1. The prefrontal cortex cannot keep enough dopamine or norepinephrine in the synapse (the microscopic gap between nerve cells where chemical signals jump across to send messages). — Genetic differences—especially in the DAT1 gene (which codes for the dopamine transporter), the DRD4 and DRD5 genes (which code for dopamine receptors), the NET gene (which codes for the norepinephrine transporter), and the COMT gene (which codes for the enzyme that breaks down dopamine)—cause the dopamine transporter to recycle dopamine too fast, or cause too few dopamine receptors (especially D4 and D5) or norepinephrine receptors (especially alpha-2A) to be built on the receiving cell, so the 'pay attention,' 'wait before acting,' and 'hold this in memory' signals fade before they finish their job or never arrive strongly enough to trigger action.
2. The attention filter fails: every stimulus—sound, thought, sight, memory, itch, worry—feels equally urgent, equally loud, and equally impossible to ignore. — Dopamine normally tags important signals (your boss calling your name, the red light ahead, the deadline tomorrow) and actively suppresses irrelevant ones (the hum of the lights, the clock ticking, a random memory from third grade). Without enough dopamine, the brain cannot prioritize, so everything floods in at once and competes for attention like a hundred browser tabs all playing sound—and you cannot close any of them or pick which one to focus on.

3. Working memory collapses: the person forgets instructions while following them, loses track of what was just said, cannot hold a phone number in mind long enough to dial it, or forgets why they walked into a room. — The prefrontal cortex needs sustained, strong dopamine signals to keep specific nerve cells firing and hold information active in your mental scratch pad for seconds to minutes. Low dopamine means new information disappears the instant attention shifts to something else—like writing on a whiteboard that erases itself the moment you look away—so you lose your train of thought mid-sentence, forget the three things you went to the store to buy, or ask the same question twice in five minutes.
4. Impulse control breaks: the person interrupts others, blurts out comments, buys things without thinking, eats impulsively, sends texts or emails they regret, or acts before considering consequences—and often does not realize they did it until after. — Norepinephrine and dopamine normally activate the 'wait and think it through' circuit in the prefrontal cortex that delays action long enough to evaluate consequences, weigh options, and choose a better response. In ADHD, that brake signal arrives too late, too weak, or not at all—so impulses become actions instantly, like a car with broken brakes rolling forward the moment you lift your foot—and the person feels shocked and regretful afterward, asking 'why did I just do that?'
5. Hyperactivity or restlessness appears: fidgeting, tapping, pacing, leg bouncing, playing with objects, an inability to sit still through meetings or movies, or a constant internal feeling of being revved up and unable to relax. — Low norepinephrine reduces the prefrontal cortex's ability to inhibit motor plans (the brain's blueprints for movement), so every small urge to move—shift position, stand up, tap a pen—becomes action immediately instead of being held back. Movement also raises alertness, norepinephrine, and dopamine slightly, so the person unconsciously moves to stay awake and keep the brain engaged—sitting perfectly still feels like fighting to stay conscious or like being trapped underwater.
6. Time blindness develops: chronic lateness, underestimating how long tasks will take, missing deadlines, forgetting appointments, losing track of hours, or feeling like time suddenly jumped forward without warning. — The prefrontal cortex organizes events along a mental timeline—what happened, what is happening now, what comes next, how much time each step needs—and uses dopamine to keep that timeline active and updated. Without enough dopamine, the internal clock stops working, the priority list dissolves, and 'now' is the only time that feels real—everything else is vague, distant 'later'—so deadlines sneak up like surprise attacks, five minutes feels like thirty seconds, and two hours vanish without a trace.

7. Emotional dysregulation: quick frustration, impatience, difficulty calming down once upset, intense reactions to minor setbacks, frequent mood swings, or feeling emotions more intensely and longer than others do. — The same prefrontal circuits that brake impulses and hold attention also regulate emotional responses—they pump the brakes on anger, disappointment, frustration, and excitement, helping you calm down and move on. Low dopamine and norepinephrine mean that emotional brake is weak or absent, so emotions escalate faster, hit harder, last longer, and feel overwhelming—like a wave that knocks you over instead of a ripple you can ride out—and others may see you as overreacting, too sensitive, or emotionally immature.
8. Functional impairment accumulates across every domain: job loss or underemployment, relationship conflict or divorce, academic failure or dropout, car accidents from distraction, financial chaos from impulsive spending or forgotten bills, legal trouble from speeding or risky behavior, or social isolation from repeated mistakes and misunderstandings. — Each broken executive function—missed deadlines, forgotten promises, impulsive decisions, emotional outbursts, chronic lateness, disorganization, unfinished projects—damages work performance, romantic and family relationships, friendships, finances, physical safety, and self-esteem. This creates a second wave of problems: shame, anxiety, depression, avoidance, substance use to cope, and a deep belief that you are lazy, broken, or defective—which makes ADHD symptoms even worse, paralyzes motivation, and traps the person in a cycle of failure and self-blame that can last for decades until the real cause is finally recognized and treated.

## Symptoms · what the patient feels, and why

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- Trouble staying focused on tasks, easily pulled away by sounds or thoughts, often makes careless mistakes: The brain needs dopamine (a chemical messenger that helps nerve cells talk to each other) to filter out distractions. In ADHD, dopamine runs low in the prefrontal cortex (the front of the brain that keeps your attention on track), so the attention system cannot ignore background noise or wandering thoughts—every little thing grabs focus and yanks it away from the task, like a radio that cannot tune out static.
- Forgets where things are, misses appointments, loses track of instructions even when trying hard to remember: Working memory (the brain's short-term notepad that holds information for a few seconds while you use it) depends on steady dopamine in the prefrontal cortex. When dopamine dips, new information slips away the moment attention shifts—like writing on a whiteboard that erases itself every few seconds.

- Acts or speaks without thinking: interrupts others, makes snap decisions, jumps into things without weighing the risks: A brake circuit in the prefrontal cortex uses norepinephrine (another chemical messenger that helps the brain stay alert and focused) to pause impulses long enough to think. Low norepinephrine means the brake is weak, so the first urge becomes an action before the person can weigh what might happen next—like a car rolling downhill with worn-out brakes.
- Restlessness or fidgeting: taps feet, shifts in the chair, feels an inner motor running all the time: Norepinephrine also helps the brain hold still by blocking extra movement signals in the motor cortex (the strip of brain that controls muscles). When levels are low, small urges to move leak out as twitches or fidgeting. Adults may feel the restlessness inside more than show it on the outside—like an engine idling too fast.
- Messy space, jumping between tasks without finishing, trouble deciding what to do first: The prefrontal cortex organizes tasks into a to-do list and ranks them by importance. Without enough dopamine, every task feels equally urgent or equally boring, so the person hops randomly instead of following a plan—like a browser with twenty tabs open and no way to pick which one matters.
- Always late, guesses wrong how long things take, misses deadlines despite wanting to be on time: Dopamine helps the brain track time passing and predict how long steps will take—like an internal clock ticking in the background. In ADHD that clock runs blurry, so minutes slip by unnoticed and the person is surprised when time runs out.
- Gets frustrated or annoyed quickly, snaps easily, takes a long time to calm down after getting upset: The same prefrontal brake that stops impulses also dials down strong emotions by sending calming signals to the amygdala (the brain's alarm center that flags danger and stirs up feelings). Low dopamine and norepinephrine mean feelings flare fast and burn longer because the brain cannot cool them as quickly—like a kettle that boils over and stays hot.
- Locks onto interesting activities so deeply that hours vanish—skips meals, loses sleep, ignores other responsibilities: High-reward or brand-new tasks flood the brain with just enough dopamine to grab full attention and make the activity feel good. But the person cannot turn that spotlight off at will, so they stay glued until the task ends or something forces a stop—like a flashlight stuck on—because the dopamine surge shuts down the brain's ability to shift gears.
- Trouble sitting through meetings or conversations, feels trapped or bored quickly, gets up to move around or finds excuses to leave: Low dopamine in the nucleus accumbens and ventral striatum (the brain's reward centers that make activities feel good or worth doing) makes slow or repetitive activities feel unbearable—like trying to sit still while someone reads the phone book. The brain craves fresh stimulation, so sitting quietly becomes physically uncomfortable and the person has to move or switch tasks to feel okay again.

- Avoids paperwork, bills, or tasks with many steps; puts them off until the last minute or past the deadline: The prefrontal cortex uses dopamine to weigh future rewards against current effort. When dopamine is scarce, distant payoffs feel invisible and the effort feels huge, so the person pushes the task away—like trying to lift a weight that feels ten times heavier than it really is. Only when a deadline creates panic does the brain get enough stress hormones like cortisol (the body's main alarm chemical) and norepinephrine to start.

## Focused exam

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- ASRS-v1.1 screener score  $\geq 14$  on the first six questions (or  $\geq 24$  on the full 18-item version): Adult ADHD happens when the prefrontal cortex (the front part of your brain that runs planning, attention, and self-control) does not get enough dopamine (a brain messenger that helps you stay focused and motivated) and norepinephrine (another brain messenger that keeps you alert and organized). The screener asks about real-life problems—losing things, forgetting appointments, starting tasks but never finishing, feeling restless. A high score flags that these troubles come from a brain-circuit problem, not just stress or bad habits.
- Collateral history from family, partner, employer, or old report cards: 'Always late,' 'loses things constantly,' 'interrupts others,' 'starts projects but never finishes': ADHD must cause real problems in at least two settings—like work and home, or school and friends. You cannot diagnose it from self-report alone, because the same prefrontal-cortex damage that causes ADHD also makes it hard to see your own patterns clearly. Hearing from people who live or work with the patient gives proof that the trouble shows up everywhere, not just in one stressful moment.
- Symptoms started before age 12 and have continued without stopping (not on-and-off): ADHD is a neurodevelopmental disorder—it comes from the way the prefrontal cortex and striatum (a deeper brain region that handles impulse control and habits) wired themselves during childhood. If symptoms only started in adulthood or come and go in episodes, the cause is something else—like depression, sleep loss, or a thyroid problem—not ADHD. The childhood-onset rule confirms the condition is built into the brain's structure, not triggered by a recent event.
- Rule out substance use (alcohol, stimulants, cannabis), obstructive sleep pauses in breathing (apnea), untreated major depression or generalized anxiety, and thyroid disease (hyperthyroidism or hypothyroidism): All of these copy ADHD perfectly—trouble focusing, restlessness, forgetting things, brain fog. Cannabis damages the hippocampus (the brain's memory recorder) and prefrontal cortex. Sleep pauses in breathing (apnea) starves the brain of oxygen at night, wrecking attention the next day. Depression drains dopamine and norepinephrine. An overactive thyroid floods your system with stress hormones; an underactive one slows everything down. If you treat ADHD without catching these first, you miss the real cause and the patient gets worse.

- Heart rate, blood pressure, and baseline ECG (a test that records the heart's electrical rhythm) are normal, especially before starting stimulant medication: Stimulants turn up the sympathetic nervous system (the 'fight or flight' wiring that speeds your heart and raises blood pressure). If someone already has heart disease, an abnormal heart rhythm, or a structural heart defect, stimulants can trigger a heart attack, stroke, or sudden cardiac death. The American Heart Association and FDA require checking the heart before starting stimulants to catch hidden risks.
- Symptoms cause measurable harm in major life areas: job loss, failing grades, broken relationships, car accidents, legal trouble—not just feeling stressed: The DSM-5-TR (the official manual doctors use to diagnose mental health conditions, published by the American Psychiatric Association) says you cannot diagnose ADHD unless the symptoms create clear, serious problems in at least two big parts of life—work, school, home, or social relationships. If someone has a few ADHD-like traits but their life is running fine, that is normal variation, not a disorder. Real harm in daily life is what separates ADHD from personality quirks.

## Diagnostics · every test answers a question

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- Thyroid-stimulating hormone (TSH) → Is thyroid disease causing the fatigue and brain fog instead of ADHD?
- Complete blood count (CBC) → Is anemia (low red blood cells) making the patient too tired to focus?
- Basic metabolic panel (BMP) → Are abnormal electrolytes or kidney trouble messing with brain function?
- Urine drug screen → Is substance use causing or worsening the attention problems?
- Electrocardiogram (ECG) → Is the heart electrically stable enough to handle stimulant medication safely?
- Baseline heart rate and blood pressure → Are there cardiovascular risks that make stimulants unsafe right now?
- Sleep study (polysomnography) if the patient snores loudly, has witnessed pauses in breathing at night, or feels exhausted despite sleeping → Is obstructive sleep pauses in breathing (apnea) (repeated airway collapse during sleep) causing the inattention and fatigue instead of ADHD?
- Structured psychiatric interview (MINI or SCID) to screen for major depressive disorder, generalized anxiety disorder, bipolar disorder, or psychosis → Are other psychiatric conditions present that need treatment before or alongside ADHD therapy?

## Treatment logic

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- ASRS-v1.1 screener plus collateral history from family, partner, or employer → This confirms the symptom pattern is present in multiple settings (work, home, social) and started in childhood, meeting DSM-5-TR diagnostic criteria before you start medication. Collateral history makes up for the fact that ADHD itself impairs self-awareness—people with prefrontal dysfunction often cannot see their own patterns accurately. External observers give you proof the diagnosis is real.
- Stimulant medication—methylphenidate (brand names Ritalin, Concerta) or amphetamine (brand names Adderall, Vyvanse)—started low and titrated (gradually increased) every 3–7 days until symptoms improve → Methylphenidate blocks the dopamine transporter and norepinephrine transporter (proteins that vacuum up these messengers after they are released), so dopamine and norepinephrine stay in the synapse (the gap between brain cells) longer. Amphetamine does that AND forces extra dopamine and norepinephrine out of storage. Both flood the prefrontal cortex and striatum with these messengers, restoring attention, working memory (the ability to hold information in your head while you use it), and inhibitory control (the ability to stop yourself from blurting things out or acting on impulse). Cochrane reviews and American Psychiatric Association guidelines show stimulants reduce core ADHD symptoms by 70–80%.
- Behavioral coaching and external structure—daily planners, smartphone reminders and alarms, time-blocking (assigning specific tasks to specific time slots), checklists, designated spots for keys and wallet → These tools offload the work of planning and remembering onto paper, apps, and the environment instead of relying on the damaged prefrontal cortex. They reduce real-world problems—missing appointments, losing things, forgetting to pay bills—while medication is being adjusted or when medication alone is not enough. The NIMH MTA study (a large National Institute of Mental Health trial) showed that combining medication and behavioral therapy beats either one alone.
- Sleep hygiene—same bedtime and wake time every day (even weekends), dark bedroom, temperature 65–68°F, no screens for 1 hour before bed, no caffeine after 2 PM → Sleep deprivation lowers dopamine receptor availability in the prefrontal cortex by 20–40%, wrecking attention, impulse control, and emotional regulation. Getting 7–9 hours per night (per CDC and American Academy of Sleep Medicine) restores dopamine receptor density, making stimulants work better and reducing impulsivity. Good sleep is rocket fuel for ADHD treatment.

- Screen and treat comorbid major depressive disorder or generalized anxiety disorder with SSRIs (selective serotonin reuptake inhibitors like sertraline, escitalopram), SNRIs (serotonin-norepinephrine reuptake inhibitors like duloxetine, venlafaxine), or cognitive-behavioral therapy (CBT, a type of talk therapy that rewires thought patterns and behaviors) → Depression and anxiety both drain dopamine and norepinephrine, and they impair the prefrontal cortex on their own. Treating these conditions restores cognitive reserve (the brain's backup capacity) and allows ADHD medication to work properly. Per American Psychiatric Association guidelines, you must treat comorbid mood and anxiety disorders at the same time as ADHD, not ignore them.
- Atomoxetine (a selective norepinephrine reuptake inhibitor, brand name Strattera) or guanfacine (an alpha-2 adrenergic agonist that activates calming receptors in the prefrontal cortex, brand name Intuniv) if stimulants fail, cause intolerable side effects, or are unsafe → Atomoxetine blocks the norepinephrine transporter, raising norepinephrine in the prefrontal cortex, which improves attention and executive function. Guanfacine strengthens the prefrontal cortex's signal-to-noise ratio—it helps you focus on what matters and ignore distractions. Both are FDA-approved for ADHD and avoid the cardiovascular risks and abuse potential of stimulants. Per American Psychiatric Association and American Academy of Pediatrics guidelines, these are second-line agents when stimulants cannot be used.
- Address social determinants of health—stable housing, reliable food, physical safety, and adequate sleep. Refer to social work, case management, or community resources as needed → Chronic stress from unmet basic needs—like not knowing where you will sleep tonight or where your next meal is coming from—activates the hypothalamic-pituitary-adrenal (HPA) axis (the body's main stress response system, which releases cortisol, the stress hormone). High cortisol damages the hippocampus and prefrontal cortex and depletes dopamine and norepinephrine. When housing, food, and safety are unstable, the brain cannot respond to ADHD treatment. Stabilizing these needs allows the brain to heal and medications to work. Per CDC and World Health Organization, social determinants are root causes of treatment failure in all chronic conditions.

## Medications

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### **Methylphenidate · Stimulant (dopamine and norepinephrine reuptake blocker)**

In adults with ADHD, two brain chemicals run too low in the prefrontal cortex (the front part of your brain that plans, organizes, decides, and stops you from acting on every impulse). Those chemicals are dopamine (controls attention, motivation, and holding back impulses) and norepinephrine (keeps you alert and focused). Methylphenidate blocks the pumps (called transporters) that normally pull dopamine and norepinephrine back into storage. When those pumps are blocked, both chemicals stay longer in the synapse (the tiny gap between nerve cells where signals jump). More chemical in the gap means a stronger, clearer signal. The

result: better focus, less impulsivity, and stronger working memory (the ability to hold information in your mind while you use it, like keeping a phone number in your head while you dial).

1. Methylphenidate enters the brain and finds the dopamine transporter (DAT) and norepinephrine transporter (NET) proteins sitting on the surface of nerve endings.
2. It binds to and blocks those transporters, like putting a cork in a drain.
3. Dopamine and norepinephrine that were just released into the synapse can no longer be vacuumed back up quickly.
4. The chemicals linger in the synapse longer, firing the receiving nerve cell more strongly and more often.
5. That stronger signal improves attention, impulse control, task initiation, and working memory within 30 to 60 minutes.

#### SAFETY WATCH

Check heart rate and blood pressure before starting and every follow-up visit; stimulants raise both. · Ask about chest pain, palpitations, or fainting—stimulants can unmask hidden heart rhythm problems. · Screen for personal or family history of sudden cardiac death, unexplained fainting, or structural heart disease; consider EKG if any red flags. · Watch for new or worsening anxiety, irritability, or insomnia—lower the dose or switch to a non-stimulant if these appear. · Ask about appetite and weight every visit; stimulants suppress hunger and can cause unintended weight loss. · Screen for substance use (especially cocaine, methamphetamine, alcohol); stimulants can be misused and should not be prescribed to someone actively using stimulants recreationally. · Ask about tics or Tourette syndrome; stimulants can make tics worse in some people. · Warn that taking the medicine too late in the day (after 4 PM for short-acting, after noon for long-acting) can cause trouble falling asleep. · Counsel on secure storage—stimulants are controlled substances and can be stolen or misused by others.

### **Amphetamine (mixed amphetamine salts or lisdexamfetamine) · Stimulant (dopamine and norepinephrine reuptake blocker and releaser)**

Amphetamines work like methylphenidate but with a second action. They block the dopamine and norepinephrine transporters (the vacuum pumps that pull these chemicals back into storage), AND they enter the nerve ending and push stored dopamine and norepinephrine OUT into the synapse (the gap between nerve cells). This double action—blocking reuptake and forcing release—floods the synapse with both chemicals, creating a very strong, clear signal in the prefrontal cortex (the brain's control center for focus, planning, and impulse control). That signal boosts attention, motivation, impulse control, and working memory. Lisdexamfetamine

(Vyvanse) is a prodrug (an inactive form) that the body slowly converts into active amphetamine in the blood, giving a smooth, long-lasting effect with lower abuse potential.

1. Amphetamine enters the brain and binds to dopamine and norepinephrine transporters on nerve endings.
2. It blocks those transporters so dopamine and norepinephrine cannot be pulled back into storage.
3. At the same time, amphetamine enters the nerve ending and reverses the direction of VMAT2 (the vesicular monoamine transporter inside the cell), dumping stored dopamine and norepinephrine into the synapse.
4. The synapse fills with both chemicals, creating a very strong signal.
5. That stronger signal sharpens focus, boosts motivation, improves impulse control, and strengthens working memory within 30 to 90 minutes (faster for immediate-release, slower and steadier for lisdexamfetamine).

#### SAFETY WATCH

Check heart rate and blood pressure at every visit; amphetamines raise both more than methylphenidate does. · Ask about chest pain, palpitations, or fainting—amphetamines can unmask serious heart rhythm problems. · Screen for personal or family history of sudden cardiac death or structural heart disease; consider EKG if any concern. · Monitor for new or worsening anxiety, agitation, irritability, or paranoia—amphetamines can trigger these, especially at high doses. · Watch appetite and weight closely; amphetamines suppress hunger more strongly than methylphenidate. · Screen carefully for current or past stimulant use disorder (cocaine, methamphetamine)—amphetamines have higher abuse potential than methylphenidate. · Ask about tics or Tourette syndrome; amphetamines can make tics worse. · Warn not to take the medicine after mid-afternoon to avoid insomnia. · Counsel on secure storage—amphetamines are Schedule II controlled substances with high street value.

### Atomoxetine · Non-stimulant (selective norepinephrine reuptake inhibitor)

Atomoxetine blocks the norepinephrine transporter (the pump that pulls norepinephrine back into nerve endings for storage) in the prefrontal cortex (the brain's control center for focus, planning, and impulse control). By blocking that pump, atomoxetine keeps norepinephrine in the synapse (the gap between nerve cells) longer, which strengthens the signal for attention and impulse control. Unlike stimulants, atomoxetine does NOT raise dopamine much, so it does NOT cause euphoria, does NOT worsen anxiety as often, and has NO abuse potential. It takes 4 to 6 weeks to reach full effect because the brain needs time to adjust its receptor sensitivity (the receiving nerve cells become more responsive to the norepinephrine that is now staying in

the synapse longer). Atomoxetine is the first choice if the person has active substance use disorder, high anxiety, tics, or cannot tolerate stimulant side effects.

1. Atomoxetine is swallowed and absorbed into the blood, then crosses into the brain.
2. It binds tightly to the norepinephrine transporter (NET) on nerve endings in the prefrontal cortex.
3. The transporter is blocked, so norepinephrine that was just released into the synapse cannot be vacuumed back up.
4. Norepinephrine stays in the synapse longer, firing the receiving nerve cell more often.
5. Over 4 to 6 weeks, the receiving nerve cells adjust by becoming more sensitive to norepinephrine, and attention, impulse control, and organization slowly improve.

#### **SAFETY WATCH**

Check heart rate and blood pressure at baseline and follow-up; atomoxetine can raise both, though usually less than stimulants. · Ask about suicidal thoughts, especially in teens and young adults—atomoxetine carries a black-box warning for increased suicidal ideation in the first few months. · Monitor liver enzymes (ALT, AST) if the person reports nausea, dark urine, yellowing of eyes, or upper belly pain—rare cases of severe liver injury have been reported. · Watch for new or worsening irritability, mood swings, or aggression, especially in the first month. · Ask about appetite and weight—atomoxetine can cause nausea and weight loss, though less than stimulants. · Warn that the medicine takes 4 to 6 weeks to work fully—many people stop too soon. · Check for use of MAO inhibitors (an older class of antidepressants)—combining them with atomoxetine can cause a dangerous spike in blood pressure. · Counsel that atomoxetine has NO abuse potential and is safe to use in people with a history of substance use disorder.

### **Guanfacine (extended-release) · Non-stimulant (alpha-2A adrenergic agonist)**

In ADHD, the prefrontal cortex (the brain's control center for focus, planning, and impulse control) is flooded with too much noisy norepinephrine signaling, which overwhelms the nerve cells and makes it hard to filter out distractions. Guanfacine activates alpha-2A receptors (a type of brake receptor) on the nerve endings that release norepinephrine. When those brake receptors turn on, they tell the nerve ending to release LESS norepinephrine, which quiets the background noise and lets the important signals come through more clearly. The result is calmer focus, better impulse control, and less emotional reactivity. Guanfacine works slowly—it takes 2 to 4 weeks to see improvement—and is especially helpful for people with ADHD plus high anxiety, tics, or trouble sleeping, because it does NOT raise heart rate or blood pressure and actually has a mild calming effect.

1. Guanfacine is swallowed and absorbed into the blood, then crosses into the brain.

2. It binds to and activates alpha-2A receptors on norepinephrine nerve endings in the prefrontal cortex.
3. Activating those receptors tells the nerve ending to release LESS norepinephrine.
4. The synapse (the gap between nerve cells) becomes quieter, with less background noise.
5. Important attention and impulse-control signals can now get through more clearly, and focus and emotional control improve over 2 to 4 weeks.

#### SAFETY WATCH

Check blood pressure and heart rate at baseline and every follow-up—guanfacine LOWERS both, which can cause dizziness or fainting, especially when standing up quickly. · Warn the person to stand up slowly from sitting or lying down to avoid lightheadedness. · Never stop guanfacine suddenly—it must be tapered slowly over at least a week to avoid rebound high blood pressure and fast heart rate. · Ask about sedation or fatigue—guanfacine can make people sleepy, especially in the first 2 weeks; taking it at bedtime can help. · Monitor for worsening depression or low mood—guanfacine can unmask or worsen depression in some people. · Avoid guanfacine in people with very low blood pressure, slow heart rate (under 60 beats per minute), or heart block. · Counsel that guanfacine takes 2 to 4 weeks to work—many people stop too soon.

### **Bupropion · Atypical antidepressant (norepinephrine-dopamine reuptake inhibitor)**

Bupropion blocks the reuptake pumps (transporters) for both norepinephrine (the brain chemical that keeps you alert and focused) and dopamine (the chemical that controls attention, motivation, and reward) in the prefrontal cortex (the brain's control center for planning and impulse control) and in deeper reward circuits (the parts of the brain that drive motivation and pleasure). By keeping both chemicals in the synapse (the gap between nerve cells) longer, bupropion strengthens the signal for attention, motivation, and mood. Bupropion is a second-line or add-on choice for ADHD—it is NOT as strong as stimulants—but it is very useful when the person also has depression, low energy, or nicotine dependence, because it lifts mood, boosts motivation, and reduces cigarette cravings at the same time. It takes 2 to 4 weeks to see full benefit.

1. Bupropion is swallowed, absorbed into the blood, and crosses into the brain.
2. It binds to and blocks the norepinephrine transporter (NET) and the dopamine transporter (DAT) on nerve endings.
3. Norepinephrine and dopamine that were just released into the synapse cannot be pulled back into storage.
4. Both chemicals linger in the synapse longer, creating a stronger, more sustained signal.

5. Over 2 to 4 weeks, attention, motivation, mood, and energy improve, and cigarette cravings (if present) decrease.

**SAFETY WATCH**

Do NOT use bupropion in anyone with a history of seizures, eating disorder (anorexia, bulimia), or abrupt alcohol or benzodiazepine withdrawal—all of these LOWER the seizure threshold, and bupropion can trigger a seizure. · Ask about seizure history, head injury, brain tumor, or heavy alcohol use before prescribing. · Start at a low dose (150 mg once daily) and increase slowly to reduce seizure risk. · Watch for new or worsening anxiety, agitation, insomnia, or irritability, especially in the first 2 weeks. · Screen for suicidal thoughts, especially in teens and young adults—bupropion carries a black-box warning like all antidepressants. · Avoid combining bupropion with MAO inhibitors—wait at least 14 days after stopping an MAOI. · Counsel that bupropion takes 2 to 4 weeks to work—many people stop too soon. · Warn that bupropion can reduce appetite and cause weight loss. · Tell the person not to crush, chew, or cut the extended-release tablets—doing so releases the full dose at once and raises seizure risk.

**Red flags**

- Attention trouble that starts for the first time after age 12 with no childhood signs—think thyroid disease (the thyroid gland in the neck controls how fast the body burns energy), sleep pauses in breathing (apnea) (breathing stops many times during sleep and starves the brain of oxygen), medication side effects, or drug use instead.
- Thoughts of suicide or deep sadness that does not lift—ADHD raises the chance of depression and acting on impulse without thinking through the outcome; check mood right away and get help urgently.
- Taking more stimulant medicine than prescribed, using someone else's pills, or mixing the medicine with alcohol or other drugs—this signals addiction risk and needs immediate help and close monitoring.
- Losing jobs over and over, getting arrested for impulsive choices, or racking up serious debt from snap decisions—the person needs close support, case management, and monitoring to prevent bigger harm.
- Active drug or alcohol problem right now—stimulant ADHD medicines (like amphetamine or methylphenidate) can make cravings worse, raise blood pressure dangerously, or trigger relapse; treat the addiction first or switch to a non-stimulant ADHD medicine like atomoxetine or bupropion.

## Nursing priorities

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1. Screen for suicidal thoughts using a direct question such as 'Have you thought about hurting yourself?' and escalate immediately to psychiatry or emergency department if the answer is yes — ADHD doubles the risk of depression and suicide because years of failure, rejection, and criticism erode self-worth; the prefrontal cortex also governs impulse control, so suicidal thoughts can turn into action faster than in other patients
2. Check blood pressure and heart rate before each stimulant dose and teach the patient to report chest pain, racing heart, or shortness of breath right away — Stimulant medications increase norepinephrine in the whole body, not just the brain, which tightens blood vessels and speeds the heart; undetected high blood pressure or heart-rhythm problems can lead to stroke or sudden cardiac death
3. Ask about sleep duration, snoring, and daytime sleepiness; refer patients with possible sleep pauses in breathing (apnea) for a sleep study before starting stimulants — Sleep pauses in breathing (apnea) causes attention problems that mimic ADHD; stimulants will not fix pauses in breathing and may worsen high blood pressure or heart strain caused by low oxygen during sleep
4. Review every medication, supplement, and over-the-counter pill the patient takes and flag any substance that slows the brain or mimics ADHD symptoms — Sedating medicines such as diphenhydramine, opioids, or benzodiazepines cloud thinking and worsen attention; treating ADHD will fail unless these medicines are stopped or switched
5. Teach the patient to take stimulant medication at the same time every morning with food and to never double a dose if one is missed — Stimulants leave the body within hours, so missed or late doses cause attention to collapse midday; doubling up raises the risk of dangerously high blood pressure, heart racing, anxiety, and insomnia
6. Help the patient set up external structure: alarms for medication and appointments, a single spot for keys and wallet, and a written daily schedule broken into 15-to-30-minute blocks — The ADHD brain cannot generate internal reminders or time awareness because the prefrontal cortex lacks enough dopamine; external cues replace the missing brain signals and prevent missed doses, lost items, and forgotten tasks
7. Screen for substance use at every visit using nonjudgmental questions and urine drug screens when appropriate; coordinate care with addiction services if use is active — ADHD raises the risk of addiction because patients may self-medicate attention problems with alcohol, marijuana, or cocaine; prescribing stimulants to someone actively using drugs increases overdose risk and medication diversion (selling or sharing prescription stimulants)

8. Document baseline weight and appetite, then recheck monthly; notify the provider if the patient loses more than 5 pounds or skips meals regularly — Stimulants suppress appetite by increasing dopamine and norepinephrine, which signal fullness; unintended weight loss can lead to malnutrition, muscle wasting, and dangerous drops in blood pressure

## Sources

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- American Psychiatric Association DSM-5-TR (2022)
- AAP ADHD Clinical Practice Guideline (2019)
- CADDRA Canadian ADHD Practice Guidelines (2020)
- CHADD evidence summaries

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