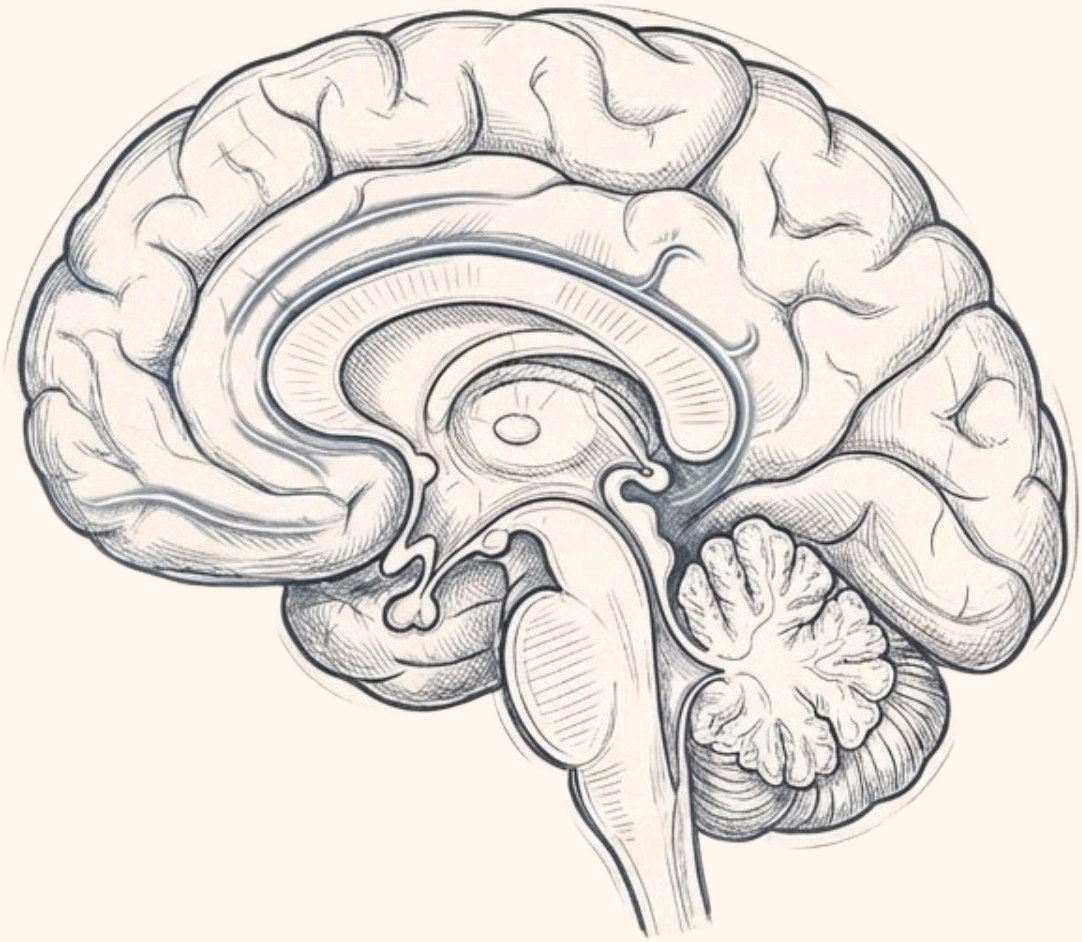
The Stuck Patient

A Guide for Therapists When Good Therapy Stops Working



Aazaz Haq, MD | McLean Neuropsychiatric Treatment Center

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Table of Contents

For The Therapist Who Is Working Harder Than the Client 1

The Lived Severity and Clinical Reality of Depression 2

Hardware and Software: A Way to Think About Why Therapy Gets Stuck 3

How to Recognize When Biology is Limiting Therapy.....5

Transcranial Magnetic Stimulation 6

Esketamine and Ketamine 8

Who Is Most Likely to Benefit 11

Closing Perspective.....13

Clinical Collaboration and Referral 14

Further Reading 17

For the Therapist Who Is Working Harder Than the Client

You have a client who shows up consistently. They want to get better. You are applying sound, evidence-based therapy. And yet, week after week, progress feels painfully slow, as if you are wading through concrete. Interventions that usually help, including cognitive behavioral therapy (CBT), eye movement desensitization and reprocessing (EMDR), dialectical behavioral therapy (DBT), or psychodynamic work, do not seem to take hold in the way you would expect.

This experience is more common than we often acknowledge. When it persists, it can quietly lead therapists to question their formulation, technique, or effectiveness, even when the work itself is thoughtful, disciplined, and clinically sound.

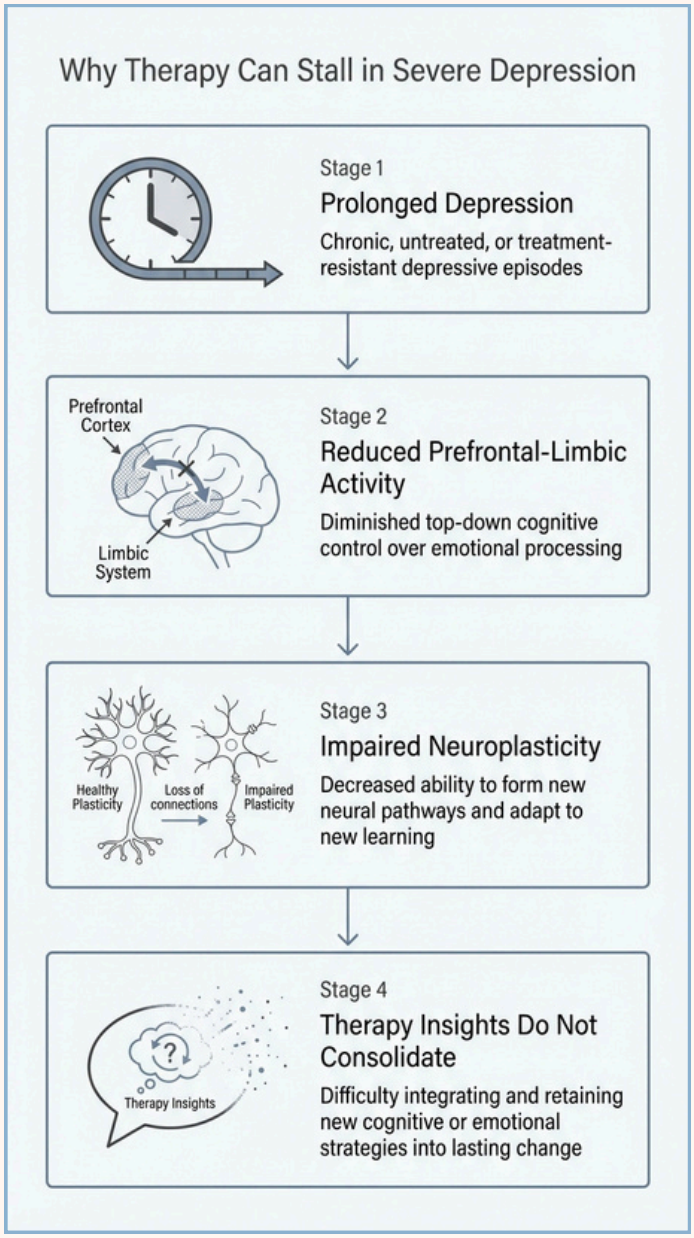
Why I Wrote This

I am Aazaz Haq, MD, an interventional psychiatrist who works almost exclusively with patients suffering from severe and treatment-resistant depression (TRD). I wrote this guide because I often encounter cases where well-conducted therapy has reached a plateau, not because the work is flawed, but because the biological severity of the illness has become a limiting factor.

In these situations, the barrier is rarely conceptual or relational. More often, it is biological. When depression reaches a certain severity or duration, the brain's capacity for learning, emotional integration, and behavioral change becomes impaired. The neuroplasticity that therapy depends on is simply not available in sufficient measure.

What This Guide Is and Is Not

This guide is not a sales pitch. It is a clinical framework designed to help you recognize when biology is limiting therapy, understand what modern interventional psychiatry can and cannot do, and decide when collaboration may meaningfully help your client.



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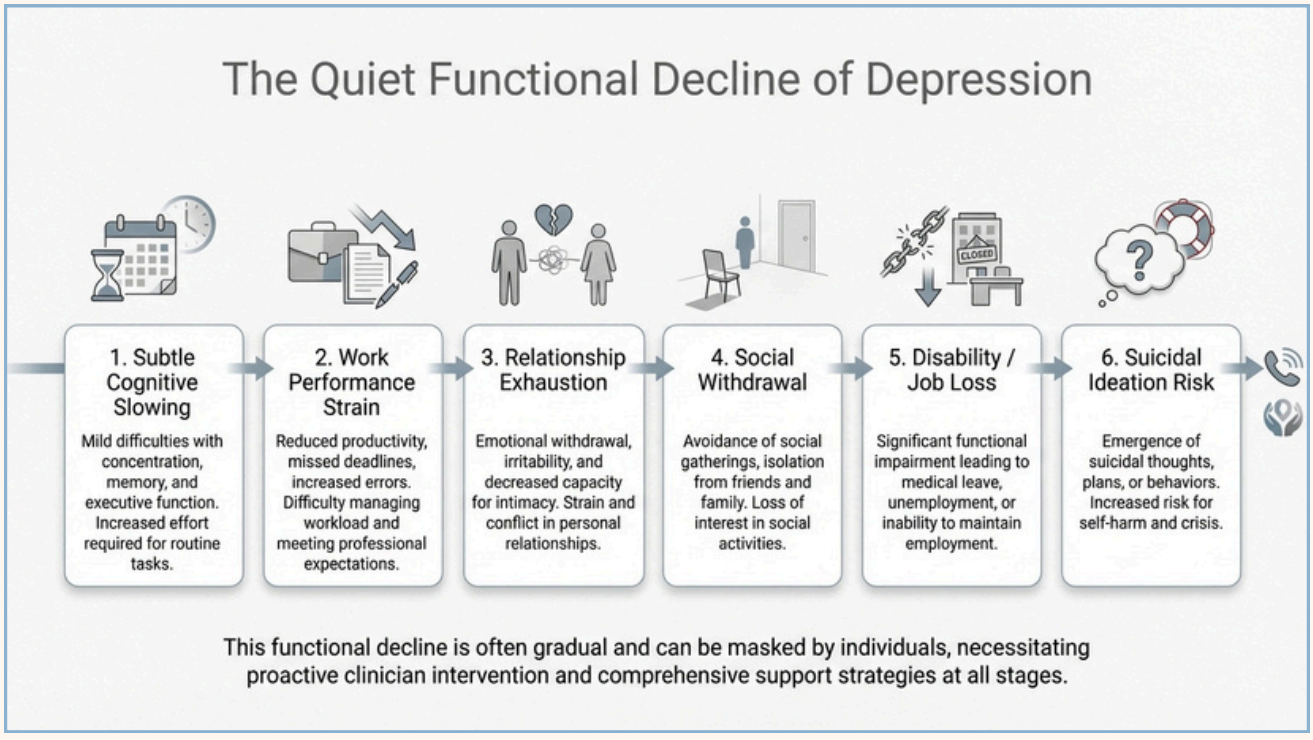
The Lived Severity and Clinical Reality of Depression

Major depressive disorder (MDD) is not only common, it is one of the leading causes of disability worldwide, and its most damaging effects often unfold quietly, over months or years, rather than all at once. Severe depression is not confined to mood. It progressively erodes cognition, motivation, physical stamina, relationships, and the ability to sustain effort in daily life.

For many patients, the course follows a familiar and tragic pattern.

Early on, depressive symptoms begin to strain personal functioning. Concentration slips. Energy declines. A spouse's concern gradually turns into exhaustion as reassurance and encouragement no longer help. Work performance becomes inconsistent, then unreliable. What once required extra effort begins to feel impossible.

Over time, functional impairment deepens. Missed deadlines at work lead to formal accommodations, then medical leave, and sometimes job loss. Social invitations are declined repeatedly until friends stop asking. Isolation increases, not by choice, but by attrition. The world quietly shrinks.



As the illness persists, internal experience often shifts as well. Passive thoughts such as "I cannot keep living like this" or "nothing will change" may give way to more active suicidal thinking. For some patients, this progression culminates in planning and intent, often unseen by those around them until risk becomes acute.

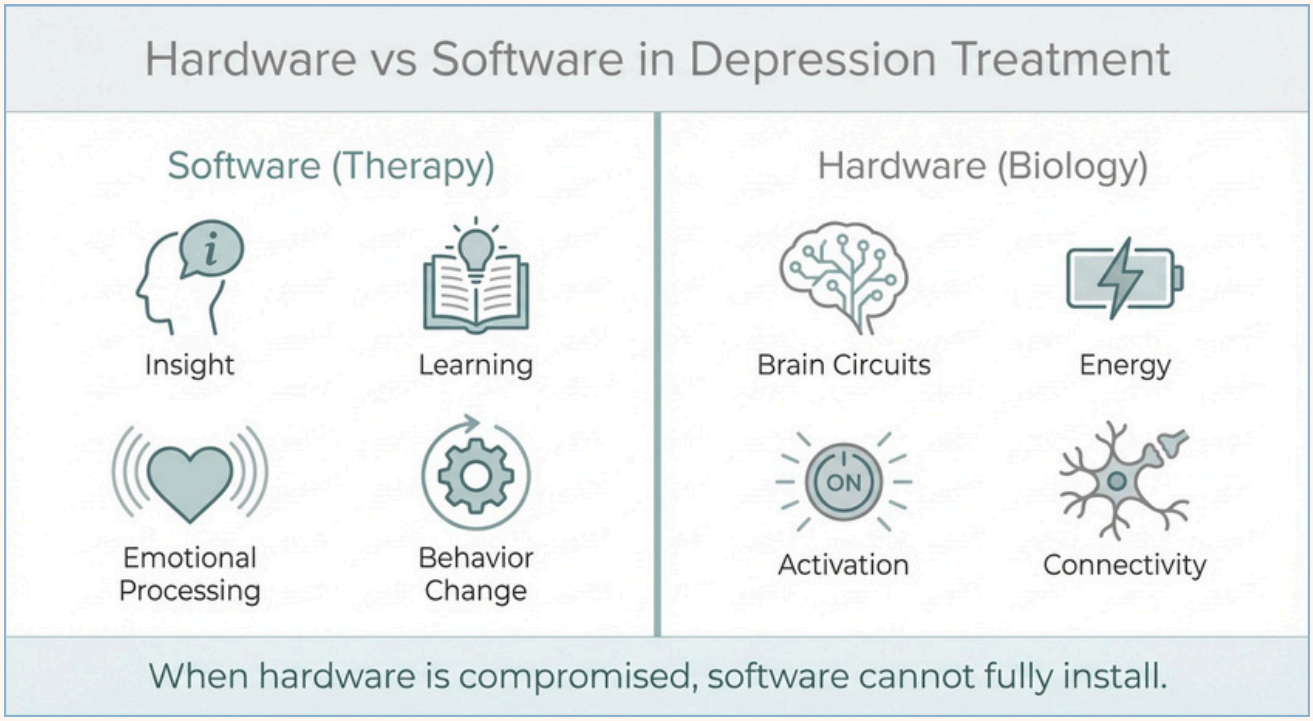
From a treatment standpoint, this trajectory is reflected in outcomes data. In the largest real-world study of depression treatment, roughly one-third of patients had still not reached remission after four treatment steps. (3) With each failed trial, hope narrows, expectations drop, and both patients and clinicians may begin to accept a diminished baseline as inevitable.

At a biological level, prolonged depression is associated with measurable changes in prefrontal-limbic circuitry, impaired reward processing, and reduced synaptic plasticity. These changes directly interfere with the brain functions psychotherapy relies on, including learning from experience, tolerating affect long enough to process it, and translating insight into sustained action.

In this context, stalled progress is not best understood as poor motivation, resistance, or insufficient therapeutic skill. It reflects the severity and chronicity of the illness itself. Without addressing these biological constraints, even excellent therapy may struggle to gain traction.

Hardware and Software: A Way to Think About Why Therapy Gets Stuck

One helpful way to think about recovery from depression is as a system with two essential parts: hardware and software. Both are important. Both are required. And difficulty in one can quietly limit the other, even when the work itself is sound.



Therapy is the software

Therapy works by introducing new information into the system. Through language, relationship, reflection, and experience, you help clients notice patterns, revise internal narratives, process painful memories, and practice new ways of responding. This is fundamentally a learning process.

For therapy to work, the brain has to be able to do several things at once. It must register new information, tolerate emotional activation long enough to stay present, compare old patterns with new possibilities, and carry insights forward into daily life. These processes rely on intact attention, working memory, emotional regulation, and the ability to initiate action.

When these capacities are available, therapy feels alive. Insights resonate rather than evaporate. Emotional experiences can be held, explored, and integrated. Behavioral experiments feel challenging but possible. The software installs because the system can support it.

Biology is the hardware

The hardware refers to the underlying brain circuits that make learning, motivation, and emotional regulation possible in the first place. In severe or long-standing depression, these circuits are often underactive and poorly coordinated, particularly in prefrontal, limbic, and reward-related networks. (6)

When the hardware is compromised, clients may understand everything you are saying and still feel unable to act. They may describe knowing what makes sense but being unable to feel it, access it, or sustain it. Everyday tasks feel effortful. Motivation does not follow intention. Emotional responses are either blunted or overwhelming.

From the outside, this can look like resistance, avoidance, or lack of engagement. From the inside, it often feels like pushing against an invisible weight. The system is operating below the threshold required for sustained change.

What this means in practice

When the hardware is struggling, even excellent therapy can feel fragile. Breakthroughs may occur but fail to consolidate. Sessions revisit the same insights without traction. Homework feels unrealistic rather than empowering. Progress depends heavily on momentary states rather than accumulating gains.

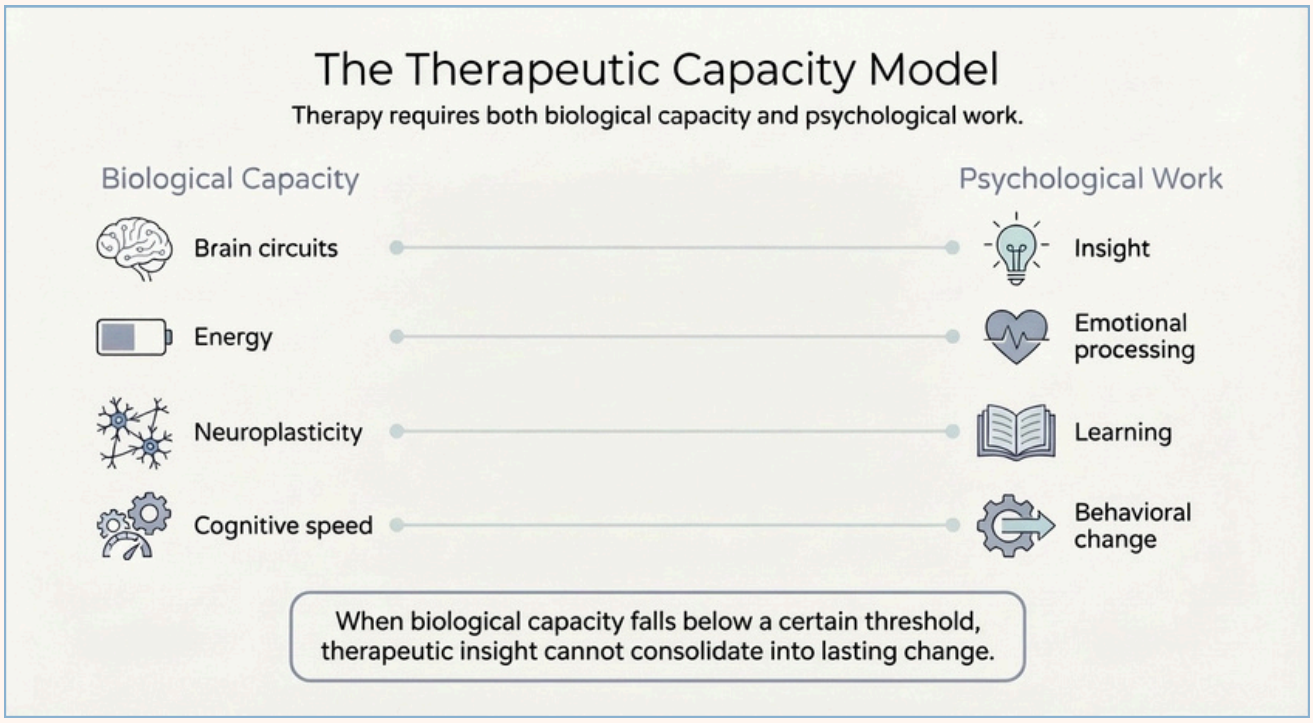
In this setting, doing more therapy or pushing harder rarely solves the problem. What is needed is additional capacity. The aim of interventional psychiatry is not to replace therapy or rewrite the software. It is to restore enough biological support so that learning, integration, and action become possible again.

Once the system has more power, therapy often changes character. Sessions feel less effortful. Emotional material becomes more accessible. Insight translates into behavior. The work you are already doing has room to take hold.

How to Recognize When Biology is Limiting Therapy

Signs therapy may be biologically constrained:

- Patient understands insights but cannot act on them
- Emotional processing does not consolidate
- Repeated cognitive insights without behavioral change
- Effort increases but capacity does not
- Severe cognitive slowing or fatigue
- Multiple antidepressant failures



Understanding Transcranial Magnetic Stimulation (TMS)

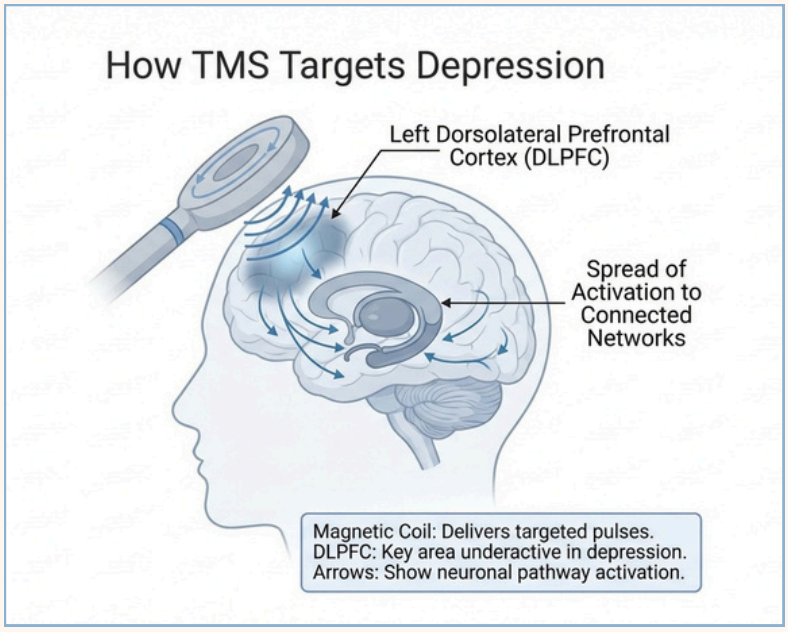
TMS is built on a simple but powerful observation: when people are depressed, certain parts of the brain are less active than they are when people are well.

Across many studies, imaging techniques such as functional Magnetic Resonance Imaging (MRI) and Positron Emission Tomography (PET) scans have shown that a specific area in the left frontal lobe, called the dorsolateral prefrontal cortex, tends to be underactive during depressive episodes. Blood flow is reduced. Glucose uptake is lower. When depression improves, whether through therapy, medication, or other treatments, activity in this region tends to increase again.

This part of the brain is important for many of the functions that depression disrupts. It plays a key role in regulating mood, keeping emotional reactions in balance, planning and decision-making, holding information in mind, abstraction, motivation, and initiating goal-directed behavior. When it is underactive, people often describe feeling mentally slowed, indecisive, unmotivated, and unable to follow through, even when they want to.

This led to a straightforward question. If a core regulatory region of the brain is underactive in depression, could directly stimulating that region help relieve depressive symptoms?

Direct electrical stimulation from outside the head is not practical. The skull blocks and disperses electrical current, and attempting to overcome that barrier would generate heat and risk injury. Magnetic fields behave differently. They pass through bone and tissue without resistance or damage. When a magnetic field changes rapidly, it induces a small electrical field in the brain tissue beneath it. That electrical field can activate neurons.



TMS uses this principle. A magnetic coil is placed over a carefully measured spot on the scalp that corresponds to the left dorsolateral prefrontal cortex. The coil generates brief, rapidly changing magnetic pulses. Each pulse induces a small electrical current in the underlying brain tissue, causing neurons to activate. When this is done repeatedly over many sessions, the brain can begin to re-establish healthier patterns of activity.

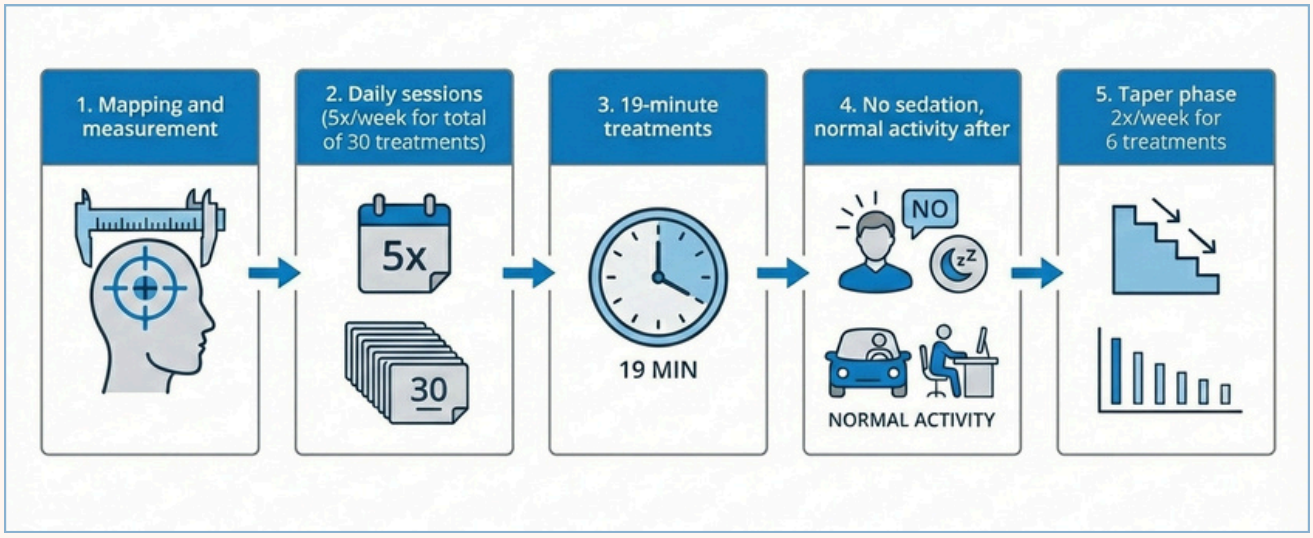
The technology behind TMS was first developed in the mid-198Os. After decades of research and refinement, it was approved by the Food and Drug Administration(FDA) for the treatment of MDD in 2OO8. Since then, treatment protocols have been optimized, targeting has improved, and outcome data have continued to accumulate. Today, TMS is widely used and supported by high-quality evidence as a safe and effective option for people with TRD.

In clinical practice, about 4O percent of patients with TRD achieve remission with TMS. Another 3O percent experience a meaningful partial response. (4) Roughly 3O percent do not experience substantial benefit. These outcomes are notable given that this population has typically not responded to multiple medication trials.

Side effects are usually mild. About 1O to 2O percent of patients experience transient headaches or scalp discomfort, most often early in treatment. The most serious potential side effect is seizure, which occurs in about 1 in 3O,OOO sessions, or roughly 1 in 1,4OO patients over a course of treatment. This is uncommon and is seen primarily in individuals with known risk factors such as a history of epilepsy or focal brain lesions.

What a TMS course actually looks like

TMS does not involve medication, anesthesia, or sedation. Patients sit comfortably in a treatment chair while a clinician takes measurements of the head to ensure precise placement of the coil. The treatment itself feels like rhythmic tapping on the scalp and is generally well tolerated.



A typical session lasts about 19 minutes. Afterward, patients are fully alert and cognitively intact. They can drive, return to work, and continue their day as usual. Treatment is usually delivered five days per week for six weeks, followed by a taper period over several additional weeks. This is the standard FDA-approved protocol for depression.

Understanding Esketamine and Ketamine

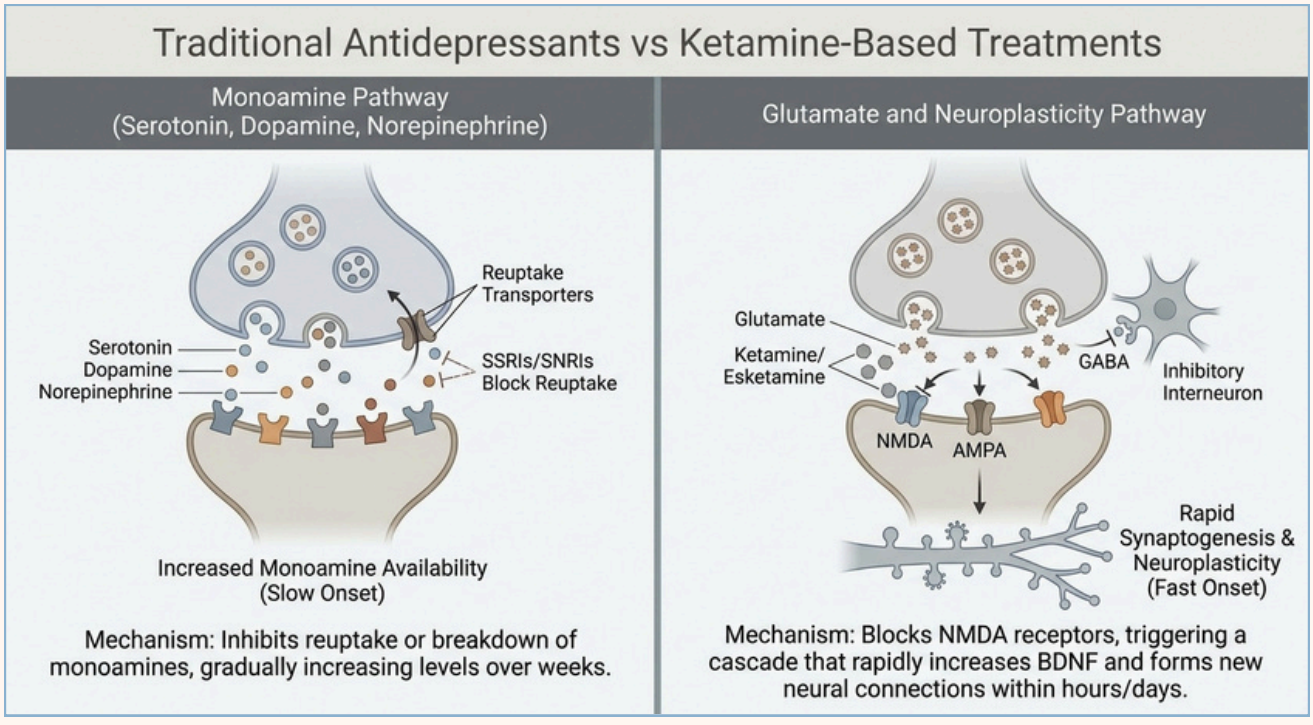
Esketamine and ketamine occupy a unique place in depression treatment because they work differently from traditional antidepressants. For many therapists, this difference helps explain why patients who have not responded to multiple medications sometimes respond meaningfully to these treatments.

A different biological pathway

Most antidepressants work through monoamine systems, primarily serotonin, norepinephrine, and dopamine. For many patients, this approach is effective. For others, especially those with long-standing or TRD, repeated monoamine-based trials yield diminishing returns.

Ketamine and esketamine act on a different system altogether. Their primary effects involve glutamate, the brain's most abundant excitatory neurotransmitter. By modulating glutamatergic signaling, these treatments appear to rapidly increase synaptic plasticity, the brain's ability to form and strengthen new connections. This represents a fundamentally different mechanism of antidepressant action.

Rather than gradually nudging neurotransmitter levels, ketamine-based treatments appear to create a temporary state in which the brain is more flexible, less rigid, and more capable of change. This helps explain why some patients who have not benefited from traditional antidepressants respond to these interventions.



Where ketamine comes from

Ketamine itself is not a new drug. It was approved by the FDA in 1970 as an anesthetic and has been used safely for decades in operating rooms worldwide. At high doses, it reliably produces unconsciousness, which is why it is also used in veterinary medicine. Because of these properties, it is a controlled substance and monitored by the Drug Enforcement Administration.

Ketamine can be misused, and esketamine carries an FDA boxed warning for abuse and misuse. That is why these treatments are given only in supervised medical settings, with screening for substance use, every dose given and monitored in the clinic, and no medication sent home.

Mirror-image molecules and esketamine

From a chemical standpoint, ketamine exists as a mixture of two mirror-image molecules, similar to how the left and right hands mirror one another. These mirror-image forms are closely related but not identical. Esketamine is one of these two forms that has been isolated and studied on its own.

In 2000, researchers at Yale University reported that a single low dose of ketamine given to patients with depression improved their symptoms rapidly, often within hours. (1) In many cases, this improvement persisted for weeks, even though the acute effects of the medication, such as sedation or dissociation, typically resolved within an hour.

What drew widespread attention was who improved. Benefits were seen not only in patients with mild or moderate depression, but also in those with severe, long-standing, treatment-resistant depression who had not responded to multiple medication trials and psychotherapy. This finding challenged long-held assumptions about how quickly and through what pathways depression could improve.

Why esketamine exists

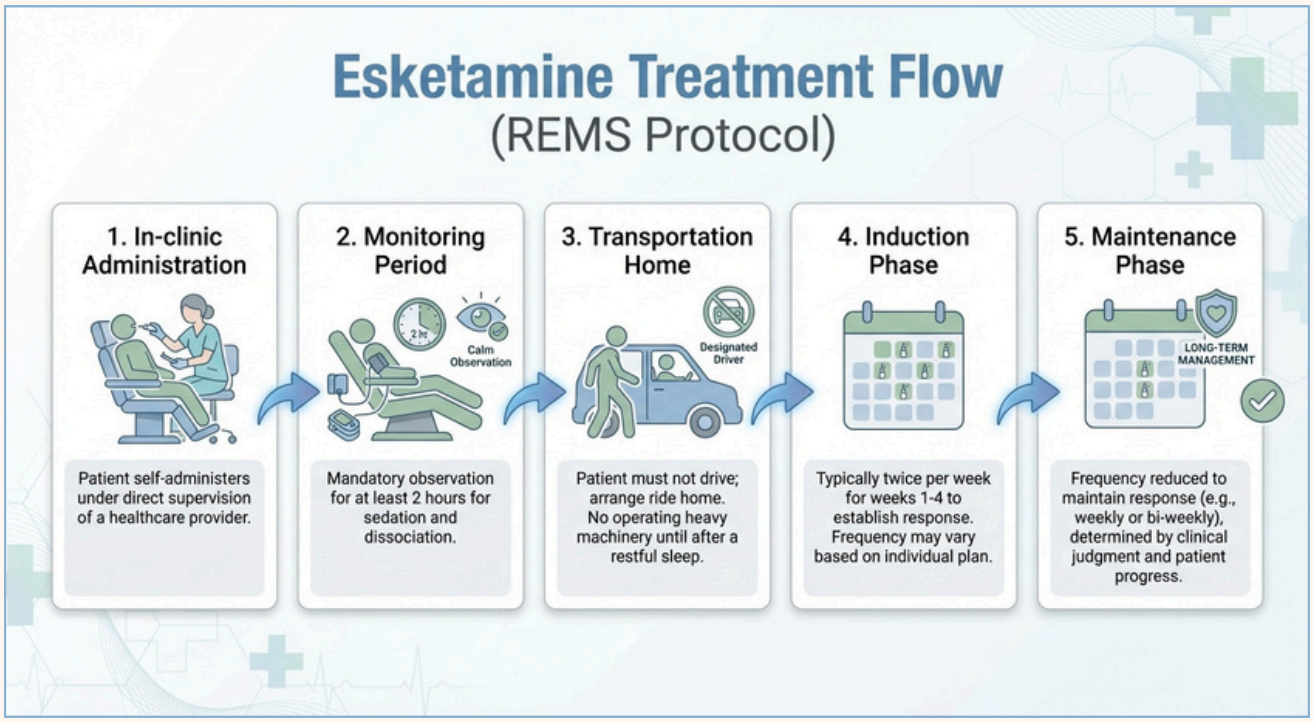
Despite growing evidence, ketamine itself has never received FDA approval for psychiatric indications. This is not due to safety or efficacy concerns, but because ketamine is a generic medication. Without patent protection, there is little financial incentive for companies to fund the large trials required for FDA approval. As a result, ketamine remains widely used off-label and is generally not covered by insurance.

Esketamine represents a practical solution to this problem. Janssen Pharmaceuticals isolated the esketamine molecule, developed it into a nasal spray called Spravato, and conducted the necessary FDA trials. In 2019, esketamine nasal spray was approved for TRD and later for depressive symptoms associated with suicidality. (2) Because of this approval, it is covered by most insurance plans, including Medicare and Medicaid.

What treatment looks like in practice

Esketamine is administered in a Risk Evaluation and Mitigation Strategies (REMS)-certified clinic under strict FDA guidelines. After dosing, patients remain in the clinic for at least two hours for monitoring. The most common side effects are sedation and dissociation, which may feel like detachment, altered perception, or a floating sensation. These effects occur in roughly half of patients, peak within the first hour, and typically resolve before discharge.

Patients cannot drive themselves home after treatment and must arrange transportation. Treatment is usually administered twice weekly for four weeks. After this initial phase, response is reassessed. Patients who benefit may continue on a maintenance schedule, often weekly or every other week, depending on clinical need.



For many patients with chronic, TRD, maintenance treatment allows sustained improvement in mood and functioning over extended periods, sometimes years. Long-term studies have not demonstrated new or cumulative adverse effects over time. At the same time, benefits may fade after discontinuation, underscoring that this is a treatment that supports recovery rather than a permanent cure.

Esketamine and ketamine are not appropriate during pregnancy, and careful counseling is essential for patients who may become pregnant.

When used thoughtfully and in close coordination with psychotherapy, ketamine-based treatments can reduce the biological rigidity of depression and create a window during which therapeutic work can deepen, consolidate, and translate into lasting change.

Who Is Most Likely to Benefit

At the most basic level, TMS and ketamine-based treatments are treatments for depression. Anyone with clinically significant depression can, in principle, benefit.

In real-world practice, access is shaped by two things.

1) Clinical fit

These treatments tend to be most helpful when depression is impairing day-to-day functioning and has not improved with standard first steps.

2) Coverage rules and logistics

Most insurance plans will only authorize esketamine or TMS for TRD after a patient has completed at least two adequate antidepressant trials, usually from different classes. Many plans also expect a trial of psychotherapy. This means that some people who would plausibly benefit may not have coverage until they have already suffered for a long time.

Where TMS fits best

TMS is FDA-cleared for MDD in patients who have not achieved satisfactory improvement from antidepressant treatment. It is also FDA-cleared for obsessive-compulsive disorder and for smoking cessation. Some devices are FDA-cleared for migraine. Certain indications require specific coils and protocols.

Beyond FDA-cleared indications, there is ongoing research and varying degrees of evidence for additional uses. Conditions with published evidence or active research include:

- Anxiety-related presentations (including generalized anxiety symptoms)
- Post-traumatic stress disorder
- Bipolar depression
- Schizophrenia symptoms (including negative symptoms and auditory hallucinations)
- Chronic pain syndromes
- Post-stroke motor recovery
- Traumatic brain injury and post-concussive symptoms
- Tinnitus
- Neurocognitive disorders and dementia-related symptoms

Evidence strength varies by condition and protocol. When we discuss off-label uses, we do so conservatively and in the context of the individual patient's presentation, goals, and alternatives.

Where ketamine and esketamine fit best

Ketamine-based treatments can be especially useful when depression is severe, rigid, and biologically entrenched, including when suicidal thinking is prominent. Esketamine is FDA-approved for TRD and for depressive symptoms in the context of acute suicidality, and it is delivered under a REMS protocol.

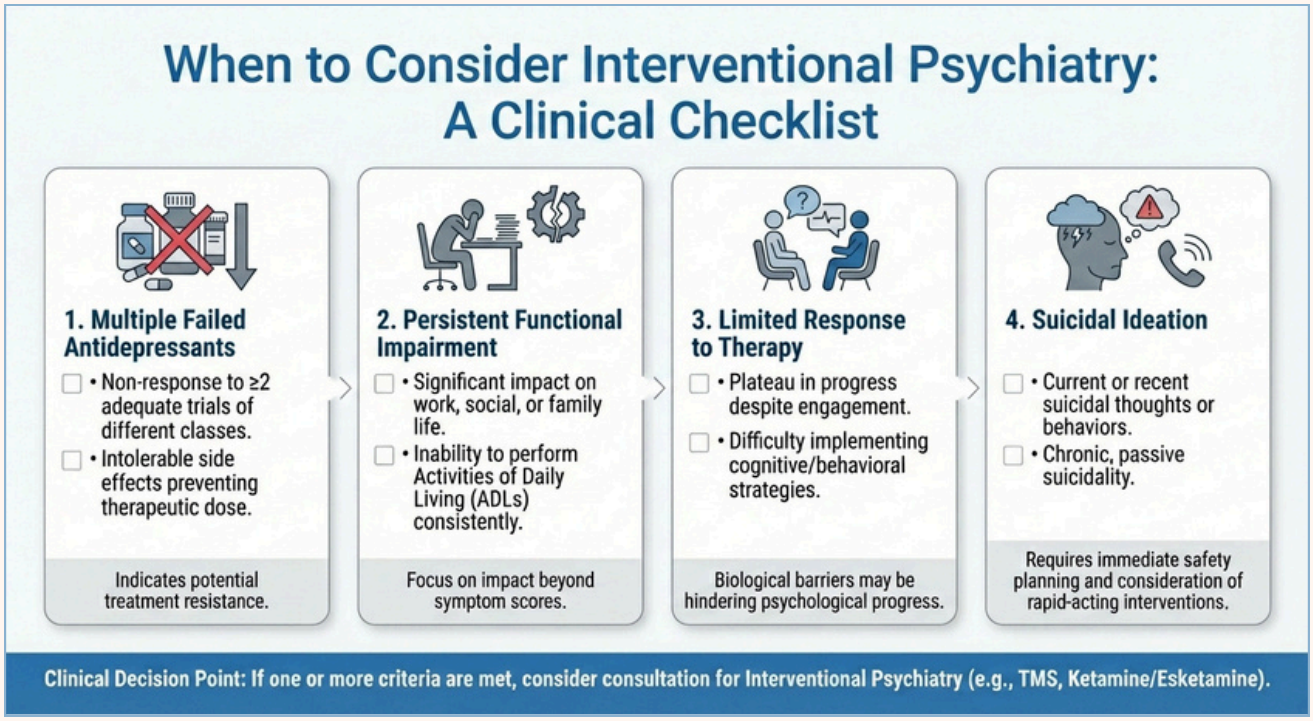
Ketamine itself is used off-label in psychiatry. In addition to depression, there is evidence supporting ketamine's potential benefit in:

- Obsessive-compulsive disorder
- Post-traumatic stress disorder
- Generalized anxiety disorder
- Substance use disorders, including alcohol use disorder and opioid use disorder, where carefully selected patients may experience reduced cravings and lower relapse risk

As with TMS, evidence strength varies by condition, and careful selection and coordination are essential.

Practical takeaway

When you are working with a depressed client who is engaged in therapy but cannot gain traction, it is often reasonable to consider biological support earlier than we have historically been trained to.



Closing Perspective

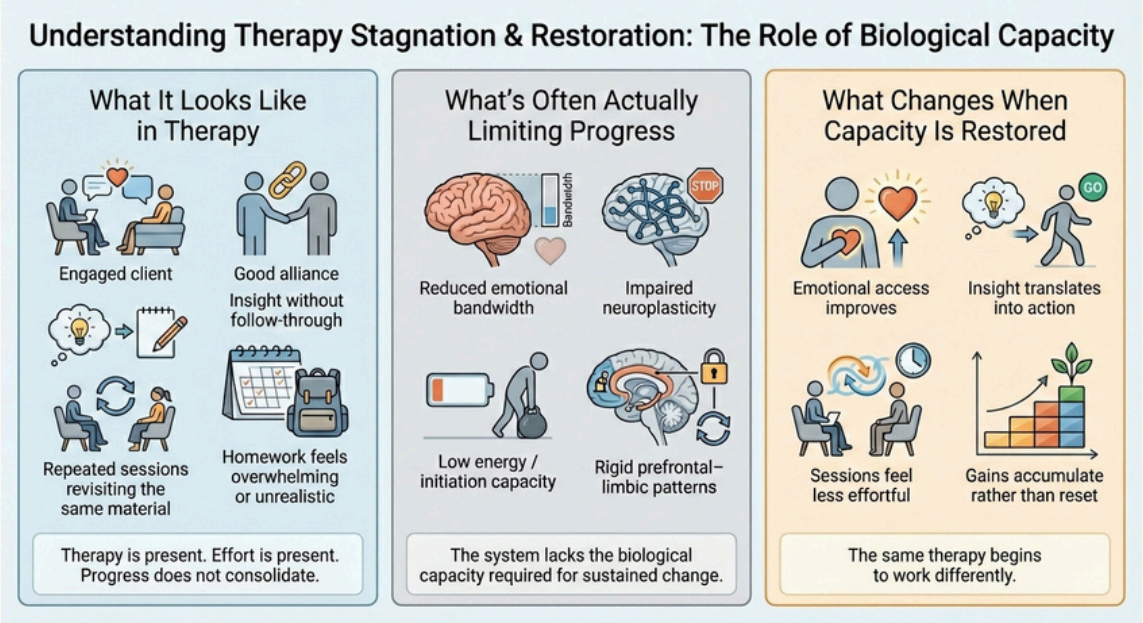
When therapy feels stalled, it does not mean the work has failed. More often, it means the biological conditions required for change are not fully in place, even when the therapeutic work itself is thoughtful, consistent, and well-executed. Naming this distinction allows clinicians to respond with curiosity rather than self-doubt, and with strategy rather than resignation.

As depression becomes more severe or prolonged, the therapy room can begin to feel narrower. Sessions take more effort, progress feels fragile, and both patient and therapist may quietly adjust expectations simply to keep going. This response is human and understandable. Over time, however, lowered expectations can harden into therapeutic pessimism, even when neither clinician nor patient truly accepts that outcome.

Interventional psychiatry offers a way to interrupt this pattern. By addressing biological constraints directly, treatments such as TMS and ketamine-based therapies can restore enough capacity for learning, emotional access, and behavioral change to occur. When this happens, therapy often changes its texture in noticeable ways. Sessions feel less effortful. Emotional material becomes more accessible. Insight is more likely to translate into sustained action.

This is not a handoff of care, and it is not a replacement for therapy. It is a collaboration. The most durable improvements tend to occur when biological treatments and psychotherapy are aligned, time-limited, and clearly integrated. (7) The therapist remains central to the work. The psychiatrist's role is to support the conditions under which that work can deepen, consolidate, and take hold.

If you are working with a client whose progress feels stalled despite sustained, thoughtful therapy, it may be worth asking whether additional biological support could help reopen the path forward. We are available to consult, collaborate, and think through these decisions together.



How Collaboration Fits Into Ongoing Therapy

By the time therapists consider interventional options, they have usually already done substantial work. The therapeutic relationship is established. The formulation is thoughtful. The client understands themselves better than they did before. What is missing is not insight, effort, or alliance.

What is missing is capacity.

In this context, collaboration with interventional psychiatry is not a referral away from therapy. It is a time-limited adjustment to the biological conditions under which therapy is occurring, with the explicit goal of allowing the existing work to deepen and consolidate.

When collaboration works well, several principles are usually present:

- The therapist remains the primary clinician and point of psychological continuity.
- Biological treatment targets severity and rigidity, not meaning or narrative.
- Roles are clearly defined, with no ambiguity about who is “treating what.”
- Communication is ongoing rather than episodic.
- The collaboration has an implicit exit plan once capacity is restored.

This is not appropriate for every client, and timing is important. In some cases, reassurance that current care is sufficient is the most clinically responsible outcome. In others, temporary biological support can meaningfully reopen a stalled therapeutic process.

The section that follows describes how we structure this collaboration in practice, with the goal of reducing burden on therapists while preserving clinical integrity and patient trust.

Clinical Collaboration & Referral

When depression is stuck, collaboration is key

Some patients do not respond adequately to standard psychotherapy and medication trials. When this happens, therapists are often left carrying the weight of persistent symptoms without clear next steps. Our role is to support you in these situations with focused, evidence-based interventional care, while preserving your central role as the patient’s therapist.

What we offer (and what we do not)

What we offer:

- Comprehensive evaluation for treatment-resistant depression
- Evidence-based interventional treatments, including TMS, esketamine, and ketamine, when clinically appropriate
- Patient education and expectation-setting around these treatments
- Management of insurance authorization and logistical barriers
- Careful monitoring of patient response and safety during treatment
- Clear communication with you at key points in the treatment course

What we do not do:

- We do not take over psychotherapy
- We do not replace the referring therapist
- We do not make unilateral treatment decisions without clinical justification

Your therapeutic relationship remains central. Our role is supportive, collaborative, and time-limited.

When a referral may be helpful

You may consider reaching out when:

- A patient has not responded adequately to multiple medication trials
- Depression remains severe or functionally impairing despite consistent therapy
- You are considering escalation but want a second clinical perspective
- You want help determining whether interventional treatments are appropriate now, later, or not at all

It is equally appropriate to decide that referral is not the right next step. Clinical judgment and timing are important.

A simple, low-friction first step

If you are unsure whether a referral makes sense, we offer an optional, no-obligation clinician-to-clinician consult.

Clinician Consult (10–15 minutes)

- Focused on one specific patient
- Case-based, not promotional
- Intended to clarify options and timing
- No requirement to refer afterward

Sometimes the most helpful answer is reassurance that continuing current care is appropriate.

How referrals work

If you and the patient decide to proceed:

- The patient contacts our clinic directly, or
- You may send a brief referral by email

We handle education, consent, and insurance processes, and we keep you informed as treatment progresses.

Our commitment to collaboration

- Respect for your clinical role and therapeutic alliance
- Clear boundaries around scope of care
- Thoughtful, conservative clinical decision-making
- Ongoing communication rather than handoff and silence

Our goal is to reduce burden on you while improving outcomes for patients whose depression has become stuck.

Next steps (optional)

If this approach aligns with how you think about care:

- Schedule a brief clinician consult to discuss a specific case
- Or refer a patient directly to our clinic

Both options are available. Neither is required.

Refer a patient or request a clinician consult

Scan the QR code or visit
mcleanntc.com/referrals

Holding hope in severe depression is not naïve. It is often a matter of having the right tools at the right moment.

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Further Reading

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