

DEPRESSION

Current theory in medicine is, "Depression is caused by low levels of neurotransmitters to include the catecholamines (dopamine and norepinephrine) and/or serotonin in the synapse." To compensate for these low levels of neurotransmitters in the synapse, physicians treat depression with SSRI medications such as Prozac, Zoloft, Paxil, and Celexa, SNRI medications such as Effexor, or Wellbutrin which according to the package insert, "...action is mediated by noradrenergic and and/or dopaminergic mechanisms."

MIXED NEUROTRANSMITTER THEORY

In a group of patients, a mixture of catecholamine and serotonin dysfunction causes depression. On one end of the spectrum are patients with a pure serotonin dysfunction. On the other end of the spectrum are patients with a pure catecholamine dysfunction. In most cases though, there is a mixture of catecholamine and serotonin dysfunction. For the system to operate optimally without symptoms requires that both the catecholamine and serotonin system are functioning properly.

"THE PROBLEM"

Other than drug side effects, patient problems encountered in the use of prescription antidepressant therapy can be divided into 2 categories:

1. Patients where the medications do not work.
2. Patients in whom the medications quit working.

The standard approaches in medicine in addressing these problems include:

1. Increase the medication dosing.
2. Change medications.
3. Add medications to the existing treatment protocol.

THE REAL CAUSE OF "THE PROBLEM"

In the Journal of Clinical Psychiatry (2000;61 Suppl 1:5-12) authors Delgado PL; Moreno FA, Department of Psychiatry, University of Arizona noted,

"NE-selective antidepressant drugs appear to be primarily dependent on the availability of NE for their effects. Likewise, 5-HT-selective antidepressants appear to be primarily dependent on the availability of 5-HT for their effects."

We have found this observation consistent with our findings:

"Drugs that work with neurotransmitters do not work if there is not enough neurotransmitters to work with."

For the patient with depression who starts treatment and finds that the drugs do not work, the problem in most cases is that the level of neurotransmitters in the brain are too low for the drugs to work.

WHY DO DRUGS QUIT WORKING?

Drugs used in depression cause neurotransmitter levels in the vesicles of the neuron (where neurotransmitters are stored) to decrease and move into the synapse. With this movement into the synapse, an increase in activity by the Monoamine Oxidase (MAO) system occurs leading to increased breakdown of neurotransmitters. Over time, further depletion of neurotransmitters can occur. When the depletion becomes great enough, the drugs quit working.

This further depletion of neurotransmitters from a clinical standpoint looks like this. A patient is started on an SSRI medication such as Zoloft for depression and initially does well for many months. The patient then literally wakes up one day to find that the Zoloft is not working any more and the depression has returned. Over time the MAO system has caused further depletion of the neurotransmitters and on the day the patient woke up to find the medications no longer working, the neurotransmitter level had just slipped below the critical level needed for the drugs to work.

BLOOD BRAIN BARRIER

Giving the patient oral, IV, or IM neurotransmitters cannot address the problem of low levels of the neurotransmitters serotonin, epinephrine, norepinephrine, and dopamine in depression. They do not cross the blood brain barrier and are unable to get to where they are needed to correct the problem.

Until now, the only way to really correct the problem was to use prescription drugs that crossed the blood brain barrier. Drugs do nothing to actually increase the number of neurotransmitter molecules in the deficiency state. Drugs work by moving the neurotransmitter molecules from one place to another in the brain, effectively tricking the brain into thinking there is more neurotransmitter molecules, when in fact, there is none.

The only other way to increase low levels of neurotransmitters in the brain is to give the patient the precursor building blocks and cofactors needed by the brain to build neurotransmitters and allow the system to correct it. The neurotransmitter precursors and cofactors cross the blood brain barrier freely where the body then turns them into neurotransmitters.

THE NEURORESEARCH APPROACH

NeuroResearch has developed a very powerful approach using neurotransmitter precursors and cofactors with laboratory testing. This approach does not merely move neurotransmitters from one place to another as drugs do. It allows the physician to follow the neurotransmitter level when giving our patented NeuroReplete in order to obtain a neurotransmitter “therapeutic range” and to truly treat and correct the problem.

TREATMENT APPROACH

For the patient with the new diagnosis of depression who is on no medications simply work the patient up properly to include thyroid dysfunction and anemia and start the patient on the NeuroReplete protocol as outlined in the "How To Treat" article.

For the patient on medications simply obtain baseline neurotransmitter tests and start NeuroReplete as per the protocol.

Once the patient is stable with therapeutic neurotransmitter levels for four to six weeks, start to slowly taper the patient off antidepressants by adjusting downward one step at a time every two weeks.

Paxil is very hard to get patients off and may require vary small milligram movements every one to two weeks near the end of weaning the patient off Paxil.

BIPOLAR DEPRESSION

Once therapeutic ranges of neurotransmitters have been established by treating the patient with NeuroReplete and RepleteExtra, as guided by neurotransmitter testing, drugs start working again or with the patient in whom the drugs did not work from the start, the drugs will work.

In our work, we have identified a small sub-grouping of patients (1% to 2%) in whom the NeuroReplete protocol still did not work. Our experience has shown that these patients are suffering from the depressive form of bipolar. Typically these patients have been depressed for years, have seen many physicians and been on many drugs without success. Once proper therapeutic ranges for neurotransmitters have been established for four to six weeks and the patient is not responding to treatment, continue using the medications and NeuroReplete and evaluate the patient for the possibility of depressive bipolar illness, and if indicated, start Lithium, Depakote or another drug used on bipolar illness. Results in our studies have been dramatic and it is our position that this is a new, unique, and valid method to assist the physician in making the diagnosis of depressive bipolar disorder.