

Management of the Effects of Neurotoxins on Neurotransmission

ON-LINE CME

Approved for 37.5
hours AMA PRA
category 1 continuing
medical education

STANDARD MEDICAL APPROACHES MAY NOT BE AS EFFECTIVE AS ONCE THOUGHT

- In several studies of reuptake inhibitors administered, only 8% to 13% of subjects obtained relief of symptoms greater than placebo.
- Treatment of the elderly (65 years and older) in the primary care setting under the monoamine theory (with reuptake inhibitors) reveals no relief of symptoms versus placebo.
Food and Nutrients in Disease Management, February 9, 2009
- The magnitude of benefit of antidepressant medication compared with placebo increases with severity of depression symptoms and may be minimal or nonexistent, on average, in patients with mild or moderate symptoms.
JAMA, January 6, 2010
- ... The true drug effect (of reuptake inhibitors) — that is, in addition to the placebo effect — was “nonexistent to negligible” in patients with mild, moderate, and even severe depression.
Newsweek, February 8, 2010
- ... more than two-thirds of patients with depression have no luck with the first medication they are prescribed.
Technology, March-April 2010

NOT JUST A BETTER APPROACH — A NEW APPROACH

This educational event is based on research driven by a database containing over 1.5 million patient days of treatment documented by over 650 clinics.

It is also based on the lecturer's research results which were published by the University of Minnesota Medical School and Johns Hopkins Medical School in 2009.

This educational event teaches a new treatment approach developed by medical doctors that is currently used by over 1,000 medical practices in the U.S.A.

PERSPECTIVE

For over 60 years, treatment of depression and other monoamine neurotransmitter diseases has been based on the monoamine theory. The use of reuptake inhibitors in clinical treatment discussed at the left is based on the monoamine theory.

The “Bundle Damage Theory” of chronic disease developed by this research project was published in peer-reviewed literature in 2009. This is the first new theory in over 60 years.

The Bundle Damage Theory recognizes chronic monoamine neurotransmitter disease causes injury to the neurons innervating and controlling function. The leading cause of neuronal injury is neurotoxic insult, although trauma and biological insult may contribute as well.

This course is dedicated to managing the cause of ongoing neuron insult along with restoring neuronal electrical flow to levels high enough to get symptoms of disease under control.

The monoamine theory predicts that getting neurotransmitter levels back to normal will control disease symptoms. The Bundle Damage Theory predicts that neurotransmitter levels need to be established higher than normal to achieve relief of symptoms.

PARTIAL LIST OF MONOAMINE NEUROTRANSMITTER DISEASES

Parkinsonism • Depression • Anxiety • Panic Attacks • ADHD • ADD • OCD • Obesity • Bulimia • Anorexia • Migraine
Headaches • Tension Headaches • PMS • Menopause Symptoms • Impulsivity • Obsessionality • Insomnia • Inappropriate
Aggression • Inappropriate Anger • Psychotic Illness • Fibromyalgia • Chronic Fatigue Syndrome • Hormone Dysfunction
• Dementia • Alzheimer's Disease • Traumatic Brain Injury • Phobias • Chronic Pain • Nocturnal Myoclonus • Irritable Bowel
Syndrome • Crohn's Disease • Ulcerative Colitis • Cognitive Deterioration • Organ System Dysfunction • Cortisol Dysfunction
• Tourette's Syndrome

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of the American Board of Quality Assurance and Utilization Review Physicians, Inc. (ABQAUWP) and a joint venture between Dr. Alvin Stein and Dr. Marty Hinz. ABQAUWP is accredited by the ACCME to provide continuing medical education for physicians.”

The American Board of Quality Assurance and Utilization Review Physicians, Inc. designates this educational activity for a maximum of 37.5 AMA PRA Category 1 Credit(s)™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

This program is approved for Category 2 credit by the American Osteopathic Association. The program is approved by the Oregon Board of Naturopathic Examiners for Continuing Education credit.

The placebo effect in treatment of depression is large. In double blind studies 30% to 45% of patients treated with placebo show resolution of symptoms.

Double-blind, placebo controlled depression studies reveal that only 7% to 13% of adult patients treated with a reuptake inhibitor get relief of symptoms better than placebo, meaning 87% to 94% of patients treated for depression with a reuptake inhibitor get no significant relief of symptoms.

Double-blind studies of elderly patients (age 65 years and older) treated with reuptake inhibitors for depression consistently reveal no treatment results better than placebo.

Not only are reuptake inhibitors marginally effective in the treatment of depression, use of reuptake inhibitors deplete neurotransmitter levels in the brain, making the very cause of the problem worse — “neurotransmitter levels that are not high enough to relieve disease symptoms”. The lowered levels of serotonin are also the logical reason for suicidal tendencies with these drugs.

Anxiety is traditionally viewed as being controlled in part by GABA, giving rise to the use of minor tranquilizers to treat anxiety. This research project has consistently demonstrated that properly balancing the serotonin and dopamine systems will give relief of symptoms, giving rise to the observation that the serotonin and dopamine systems control GABA.

It is an observation of this research project that problems and side effects that occur in treatment of Parkinson's patients with L-dopa preparations are caused by mismanagement of the amino acid L-dopa. L-dopa must be given in proper balance with serotonin precursors along with sulfur amino acids in order to manage problems properly.

THE BUNDLE DAMAGE THEORY AS PUBLISHED

Neurotransmitter dysfunction disease symptoms, such as symptoms of depression, develop when the electrical flow through the neuron bundles that regulate function is compromised by damage to the individual neurons or the neuron components composing the neuron bundle which conducts electricity to regulate or control function. In order to optimally restore neuron bundle regulatory function, synaptic neurotransmitter levels of the remaining viable neurons must be increased to levels higher than is normally found in the system. This restores adequate electrical outflow resulting in relief of symptoms and optimal regulatory function.

PARADIGM
Shift

Low levels of neurotransmitters in the brain are not the cause of the majority of cases of chronic monoamine neurotransmitter diseases listed on at the bottom of page 1. Chronic monoamine neurotransmitter disease is caused by damage to the neurons that conduct electricity in the brain. Review of over 100,000 monoamine assays in patients prior to treatment reveals no significant difference

in patients suffering from or not suffering from disease.

Laboratory assay of monoamine neurotransmitters is not an assay of the neurotransmitter levels of the body. The levels of monoamines found on assay are a function of transporter status. Proper assay with analysis of monoamine neurotransmitter levels reveals the functional status of organic cation transporters. Optimization of transporter function is key to achieving optimal results in treatment. In monoamine disease states, low levels of neurotransmitters are not the etiology. Neurotransmitter levels that are not high enough to compensate for compromised electrical flow, due to neuronal damage is the cause. Elevating monoamine neurotransmitter levels higher than normal must be done in a way that achieves proper balance between the serotonin and catecholamine systems.



Drugs do not increase the number of monoamine neurotransmitter molecules in the brain. They work by moving neurotransmitters from one place to another and in the process set up conditions that deplete monoamine neurotransmitters. In this course we teach National Institute of Drug Abuse model explaining how reuptake inhibitors deplete neurotransmitters. The only way to truly increase the

total number of monoamine neurotransmitter molecules in the brain is through administration of properly balanced amino acid precursors which cross the blood-brain barrier and then are synthesized into new neurotransmitters.

THE COURSE

In 2009, the University of Minnesota Medical School published the first of a series of papers it is writing on the findings of this research project. Also in 2009, Johns Hopkins Medical School published findings of this research project.

The leading cause of chronic monoamine neurotransmitter disease is not from low levels of neurotransmitters, it is from damage to neurons.

This course provides a comprehensive education on the:

1. Identification and management of neurotoxins.
2. Proper treatment required to compensate for the decreased flow of electricity in the brain caused by neuronal damage from neurotoxins.
3. Interpretation of monoamine assays in the clinical setting, in order to optimize organic cation transporter function with associated relief of symptoms.

ON-LINE COURSE OVERVIEW

Cutting-edge research approved for 37.5 hours of AMA category 1 CME

For a comprehensive overview and full course objectives go to: www.NeuroSupport.com

MANAGEMENT OF MONOAMINE NEUROTRANSMITTER DISEASE

MODULES 1-6 / 15.5 HOURS

The participant will understand and be able to do the following:

1. Understand synthesis and regulation of serotonin and catecholamines.
2. Understand how the serotonin system and catecholamine system function as one system with focus on synthesis, metabolism, and uptake.
3. Understand and differentiate between the bundle damage theory and monoamine theory.
4. Be able to generate an extensive list of diseases caused by or associated with monoamine neuron dysfunction.
5. Understand the role of monoamines as neurotransmitters and neurohormones in regulatory functions, autocrine functions, and paracrine functions.
6. Under the National Institute of Drug Addiction model, understand how reuptake inhibitors deplete monoamines and how to compensate for this safely.
7. Understand how improperly balanced amino acid precursors of the serotonin and catecholamine system deplete neurotransmitters.
8. Understand proper use of L-dopa with serotonin precursors and sulfur amino acids to optimize treatment results in Parkinson's disease and other monoamine illnesses.
9. Understand basic physiology of kidneys, peripheral system and central nervous system with regard to monoamines.
10. Understand the three-phase model of urinary monoamine analysis leading to determination of organic cation transporter status.
11. Be able to perform basic interpretation of urinary monoamine assays in order to determine and optimize organic cation transport.
12. Understand the efficacy of reuptake inhibitors in treatment of depression.
13. Understand the monoamine optimization approach to depression as a basis for treating other illness.
14. Understand and implement treatment protocols in the treatment of monoamine disease.
15. Understand and manage common side effects associated with amino acid precursor administration.
16. Understand the importance of sulfur amino acids and their proper dosing in treatment.
17. Understand the tyrosine base to prevent dopamine fluctuations.
18. Understand special considerations in the treatment of various monoamine-related diseases.

MANAGEMENT OF NEUROTOXINS

MODULES 7-13 / 14 HOURS

The participant will understand and be able to do the following:

1. Understand the definition of neurotoxin and be able to understand the difference between acute and chronic poisoning.
2. Understand the OSHA requirements for toxin identification as it relates to clinical practice.
3. Define "body burden" and discuss how it can be a problem.
4. Describe how body burden is developed, what it is comprised of, and how it affects both adults and children.
5. Understand how everyday products we consume, clean our bodies and homes with, and things we find in our home environment may be toxic and have adverse effects on our health.
6. Generate a list of the more common solvents encountered to include how they are used and how they affect the body.
7. Define what a pesticide is, explain the difference between active and inert ingredients, identify the different pesticide classes and identify pesticides that fall within those classes.
8. Describe the toxic effects of pesticides on adults and children.
9. List the pesticides most persistent in the environment and give details about each of these.
10. Identify random toxins that do not fit any specific category and list their effects.
11. Identify typical genetically modified foods.
12. Understand ramifications of genetically altering our plants from ethical and scientific perspectives.
13. Understand the effects of genetic modification along with the results.
14. Understand the effects of genetic modification on health.
15. Define heavy metals, explain how they become toxic, and identify the seven metals that are most neurotoxic.
16. Understand where the seven most toxic heavy metals are found in the environment, how human exposure occurs and potential impacts on health.
17. Understand treatments for heavy metal exposure.
18. Identify lab test deviations from normal that identify toxic substances and interpret these results.
19. Successfully detoxify patients suffering from environmental toxin exposure.



CME
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877-626-2220
www.NeuroSupport.com

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OBESITY: A MONOAMINE DYSFUNCTION DISEASE

Diseases and states that occur secondary to obesity:

- Decreased life expectancy
- Type 2 diabetes
- Heart disease
- Increased risk of stroke
- Sleep apnea
- Knee problems
- Back problems
- Increased rehabilitation time
- Increased rate of injury
- Increased risk of colon, uterine, and breast cancer
- Renal failure
- Vascular disease
- Increased risk of gallstones
- Gynecological irregularities
- Female fertility problems
- Hypertension
- High cholesterol
- Hiatal hernia
- Increased risk of lung infections
- Increased risk of gastric ulcers
- Fibromyalgia
- Myoclonus

MODULES 14-16 / 8.0 HOURS

The participant will understand or be able to do the following:

1. Understand how serotonin and norepinephrine control the appetite center of the brain.
2. Understand various pharmacologic and non-pharmacologic approaches for inducing appetite suppression through monoamine (serotonin / norepinephrine) manipulation.
3. Understand approaches in evaluating patients to determine if the appetite is under adequate control to allow patients to eat less food comfortably and lose weight.
4. Evaluate the patient at clinic visits to determine whether the patient is on track to make goal weight and take corrective action as needed.
5. Understand and manage the impact of motivation and time between clinic visits on successful weight loss.
6. Understand the impact of caloric intake on time to goal weight and successful weight loss.
7. Understand the impact and use of computers in evaluating weight loss to determine whether patients are on track to make goal weight or corrective action is needed.
8. Understand strategies for safely stopping insulin and oral hypoglyemics during effective weight loss.
9. Understand management of secondary disease processes as they resolve during effective weight loss.

For comprehensive overview and complete course objectives go to: www.NeuroSupport.com

REGISTRATION

Call 877-626-2220

On-line registration is not available.

QUESTIONS

E-mail us: CME@NeuroSupport.com

OBTAINING HOURS

The course is comprised of 16 modules.

Each module is comprised of 2 to 5 slide shows with voice narrative ranging in length from 15 minutes to 2 hours. At the end of each slide show is a post test. When the post test is completed successfully credit will be given for completing that slide show. When all slide shows in a module are completed CME credit will be given for the number of hours comprising that module.

When the participant indicates in writing that course work is finished a certificate for the number of hours completed will be issued.

TECH SUPPORT

Pre-course on-line orientation with simultaneous over-the-phone instruction is provided. Full tech support during the course is provided via phone and e-mail.

FACULTY

Marty Hinz, MD and Alvin Stein, MD