MEDICAL CONFERENCE

Clinical Management of Centrally Acting Monoamine and/or Thiol Deficiency Conference

The foundation of this conference is 22 peer-reviewed papers authored by the lecturers.

Course Directors:

Marty Hinz, MD
Stephen Center, MD
Ross Stewart, PhD

Los Angeles

Presenting peer-reviewed original research papers that were co-authored with faculty at the University of Minnesota Medical School in Duluth, MN

NeuroResearch
A Medical Education Company

Friday April 27, 2018 -- 9 AM
until
Sunday April, 2018 -- Noon

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Physicians: This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of AKH Inc., Advancing Knowledge in Healthcare and NeuroResearch Centers, Inc. AKH Inc., Advancing Knowledge in Healthcare is accredited by the ACCME to provide continuing medical education for physicians. AKH Inc., Advancing Knowledge in Healthcare designates this live activity for a maximum of 15 *AMA PRA Category 1 Credit(s)™*. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Identification and Management of Disease Associated Centrally Acting Monoamine and Thiol Deficiencies:

Centrally acting monoamines and thiols are naturally occurring substances found in the body.

The **CENTRALLY ACTING MONOAMINES** are:

1. Serotonin
2. Dopamine
3. Norepinephrine
4. Epinephrine (adrenaline)

The **THIOLS** are:

1. L-methionine
2. S-adenosyl-methionine
3. S-adenosyl-homocysteine
4. Homocysteine
5. Cystathione
6. L-Cysteine
7. Glutathione

PERSPECTIVE

While there is a Saturday segment of the conference dedicated to the management of obesity and associated illnesses, the primary focus of this conference is not exclusively dedicated to obesity or a specific disease. It is dedicated to serotonin, dopamine, norepinephrine, epinephrine, and thiol optimization techniques as documented in the peer-reviewed medical literature.

THE ROOTS OF THIS RESEARCH

In the early 1990s, union members in Duluth, MN demanded the benefit of medical insurance coverage for obesity treatment. With this, Duluth became the only community in the United States where treatment of obesity (BMI >30 with no other risk factors), was viewed as a medical risk factor and covered by insurance, to include Blue Cross. By 1994, the Morgan Park Medical Clinic was the only clinic based medical weight loss program reimbursed by all commercial medical insurances in the United States. The other Duluth program we worked closely with, was hospital based.

The Morgan Park Clinic operated as a general medical clinic where obesity was treated just like any other risk. It was common to simultaneously see diverse patients such as a weight patient, a cast removal, and a chest pain that needed stabilization and transfer to the local hospital. After each patient visit we databased everything possible. Not being hindered by the patient's financial resources, generated a treasure trove of observations and data not possible with any other approach.

APPETITE SUPPRESSION

The most effective way to lose weight is by eating less food. If the patient is extraordinarily hungry it is difficult to eat less food. The appetite center of the brain is controlled by norepinephrine and serotonin as evidenced by the fact that drugs in the pharmacologic class known as anorectics work only with norepinephrine and/or serotonin.

As patients were placed into appetite suppression, using methods approved by the FDA and/or Minnesota State Medical Board. It soon became apparent that the average patient was also suffering from three or more other diseases listed on the right side of the yellow box page 3. As serotonin and catecholamines were manipulated for optimal appetite suppression, symptoms of these other diseases usually resolved. What started out as insurance reimbursed treatment of obesity turned into an effective and comprehensive treatment for the symptoms of other diseases associated with neurotransmitter (serotonin and/or catecholamine) disease.

THIS CONFERENCE

It has been 20 years since treatment of obesity and other diseases were unified. Success is more than writing a prescription, patient support and positioning is critical. Even if you don't treat obesity as a risk factor, the techniques taught in this conference are appropriate for all patients suffering from disease relating to serotonin, catecholamines, and thiols.

Serotonin and catecholamine optimization is based on database statistical analysis:

Serotonin, Dopamine, Norepinephrine, and/or Epinephrine Deficiencies:

Presenting peer-reviewed original research papers that were co-authored with faculty at the University of Minnesota Medical School in Duluth, MN

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Data from over 2,000 clinics
THIS RESEARCH IS BASED ON STATISTICAL ANALYSIS OF:

1. **The Other disease database**
   -4 million+ patient-days of care
2. **The Parkinson's disease database**
   -Data from over 1,450 patients
3. **The Neurotransmitter lab database**
   -2.5 million+ patient days of care
4. **The Weight loss database**
   -3 million+ patient days of care

Upon hearing of these databases, the University of Minnesota Medical School in Duluth, Minnesota entered into an agreement to co-author research results. Since then, 22 peer-reviewed papers that have been published will be presented.

The scope and complexity of these databases cannot be overstated. It is envisioned that these databases and their statistical analysis will be generating peer-reviewed research data for many years to come.

Serotonin and/or catecholamine deficiency associated or induced conditions (partial list)

OBESITY

Decreased life expectancy
Diabetes (Type II) → **Type II DIABETES (Goes away with weight loss)**
Heart disease
Increase incidence stroke
Sleep apnea
Knee problems
Back problems
Increased rehabilitation time
Increased rate of injuries
Female fertility problems
Gynecologic irregularities
Gouty arthritis
High blood pressure → **HYPERTENSION HYPERCHOLESTEROLEMIA**
Hiatal hernia
High cholesterol → **HYPERTENSION HYPERCHOLESTEROLEMIA**
Increased lung infections
Increase in gastric ulcers
Chronic pain
Fibromyalgia
Myoclonus

Type II DIABETES (Goes away with weight loss)
Decreased life expectancy
Increased infections
Diabetic neuropathy
Kidney failure
Macular degeneration
Heart disease
Foot ulcers
Therapeutic amputations
Disability
Increase incidence of stroke
Impotence

HYPERTENSION HYPERCHOLESTEROLEMIA
Decreased life expectancy
Heart disease
Stroke
Kidney failure
Vascular disease
Ischemia

INCREASED CANCER RISK
Increased colon cancer
Increased uterine cancer
Increased breast cancer

ALL the problems listed above are caused by or exacerbated by eating too much food (obesity).

At the heart of all these diseases is obesity (eating too much food). The only way to lose **significant** weight is to eat less food (decreased calorie intake). Serotonin and norepinephrine control the appetite center of the brain. The only drugs which control appetite and allow the patient to eat less food comfortably are the anorectic class of drugs in pharmacology. All of these drugs are serotonin and/or norepinephrine reuptake inhibitors.

OTHER DEFICIENCY DISEASES

DOPAMINE DOMINANT ETIOLOGY
Parkinson's disease
Restless Leg Syndrome

NEEDS DIFFERENTIATION
Adrenal fatigue
Alcoholism
Allergies / histamine driven
- Allergy induced asthma
- Multiple chemical sensitivities
- Peanut or other food allergies
- Urticaria, chronic recurrent

Bipolar
Essential tremor
- Rule out Parkinson's disease

Fatigue (negative metabolic workup)
GI disorder:
- Crohn's
- Irritable bowel disease
- Ulcerative Colitis

Hormone dysfunction
- Cortisol dysfunction
- Premenstrual Syndrome (PMS)

Lyme disease
Psychotic illness
Schizophrenia

SEROTONIN DOMINANT ETIOLOGY
Addiction
Alzheimer's (dementia)
ADD
ADHD
Autism

Cognitive deterioration
Chronic neurotransmitter depletion:
- Chronic illness
- Chronic pain
- Chronic stress

Depersonalization disorder
Depression
Eating disorder (anorexia / Bulimia)
GABA dysfunction
- Anxiety
- Glutamate regulation
- Panic disorder (attacks)
- Stiffman Syndrome

Hyperactivity
Insomnia
Obsessive Compulsive Disorder (OCD)
Organ system dysfunction
Phobias
Post-traumatic stress disorder (PTSD)
Seasonal affective disorder
Social anxiety disorder
Serotonin driven cardiac disease
Tension headaches
Tourette's Syndrome
Traumatic brain injury
Trichotillomania
Fibromyalgia
Migraines
- Abdominal
- Headache
- Atypical

NeuroResearch Centers
877-626-2220
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The focus of this conference is not obesity or a specific disease, it is dedicated to serotonin, dopamine, norepinephrine, epinephrine, and thiol general optimization techniques.

These two pages (pages 4 and 5) are a small sampling of the numerous topics presented at the conference.

THE OTHER DISEASES

Examine the right side of the yellow area on page 3. At some point in the history of medicine, a caregiver has prescribed reuptake inhibitors for each of these diseases. The initial problem we encountered was anorectic (norepinephrine reuptake inhibitor) drugs that quit working soon into treatment. Eventually, we found solutions that applied to all reuptake inhibitors.

NIH-NATIONAL INSTITUTE OF DRUG ABUSE

Then, National Institute of Drug Abuse model documents the mechanism of action whereby reuptake inhibitors deplete neurotransmitters. When neurotransmitter depletion becomes too great, the drugs quit working. Literature notes, "Reuptake inhibitors appear to be primarily dependent on the availability of neurotransmitter for their effects."

STATISTICAL ANALYSIS

After extensive multiple variable database analysis, methods for effectively preventing or compensating for the impact of reuptake inhibitor-induced neurotransmitters depletion were refined.

COMPREHENSIVE REVIEW

The speakers will present their papers discussing how reuptake inhibitor depletion has been linked to the following and comprehensively review the known options available for effective management of each:

1. Low efficacy (only 7% to 13% of depressed patients treated with reuptake inhibitors achieve results better than placebo 30-days into treatment)
2. Placebo relapse
3. Suicidal ideation
4. Symptom relapse
5. Discontinuation syndrome
6. Drug tachyphylaxis in general

DIFFERENTIATION

Many diseases listed in the yellow area of page 3 are not exclusively caused by serotonin dysfunction or dopamine dysfunction. This conference will teach strategies for clinically differentiating serotonin dysfunction from dopamine (catecholamine) dysfunction to facilitate optimal initiation of care.

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PARKINSON'S DISEASE

Even if you are not currently treating Parkinson's disease patients, everyone should be aware of the following. Parkinson's disease is a prototype disease for the study of all dopamine (catecholamine) related problems. While other dopamine diseases may have some of the problems associated with dopamine management, Parkinson's disease has them all.

THE MOST EFFECTIVE TREATMENT

The most effective Parkinson's treatment is the drug L-dopa, which crosses the blood brain barrier then is freely metabolized to dopamine without biochemical feedback regulation. 89% of Parkinson's disease patients take the drug combination L-dopa/carbidopa.

THE DRUG L-DOPA

Chronic administration of the drug L-dopa may cause depletion of serotonin and all seven thiols. Speakers at this conference, will present peer-reviewed papers documenting how the standard approach of administering L-dopa/carbidopa has been linked to 29 systemic depletions which affect hundreds of enzymes and proteins. At this conference peer-reviewed strategies for depletion identification and management will be presented.

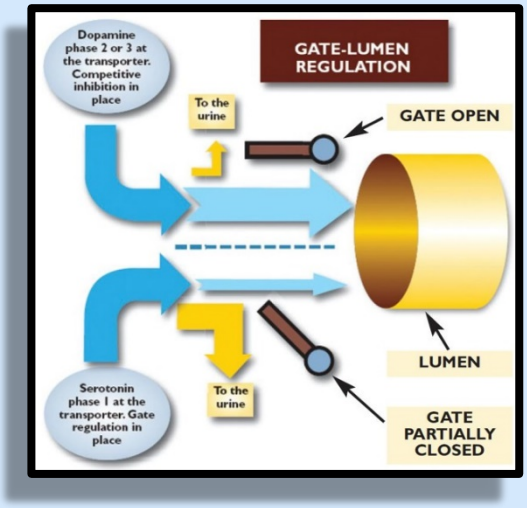
CARBIDOPA

Carbidopa is of no value in treating Parkinson's disease symptoms. Its only indication is control of L-dopa-induced nausea. Authors of recent original papers presented at this conference will present published research which identifies:

1. The mechanism of action and effective management when the worsening symptoms of Parkinson's disease are caused by the reversible carbidopa-induced effects.
2. The mechanism of action linking carbidopa to the 400%+ increase in the Parkinson's death rate since 1975 published by the Center for Disease Control.
3. The mechanism of action and effective management of carbidopa-induced L-dopa tachyphylaxis.
4. The mechanism of action and effective management of carbidopa depleting serotonin, GABA, and histamine secondary to interference with synthesis.
5. The mechanism of action when carbidopa compromises function of over 300 enzymes and proteins.
6. The mechanism of action and effective management of carbidopa-induced dyskinesias and choreiform movements previously thought to be caused by the drug L-dopa.
7. The 100 most common side effects caused by carbidopa.
8. The published mechanism of action and effective management of on/off effect.

These two pages (pages 4 and 5) are a small sampling of the numerous topics presented at the conference.

LABORATORY OPTIMIZATION



Control of intercellular and extracellular serotonin and dopamine concentrations, to include synaptic concentrations, is ultimately the responsibility of the organic cation transporters type-2 (OCT-2).

Papers will be presented which outline approaches to determining OCT-2 functional status as related to serotonin and dopamine concentrations of the body.

Original data demonstrating how the functional status of the OCT-2 transporters correlates with serotonin and dopamine concentrations in response to numerous types of external stimuli will be presented.

MEDICAL WEIGHT LOSS LECTURE

Weight loss data:
Average weight loss first 30-days = 16.9 lbs
83% female, average starting weight 211 lbs, average goal weight 143 lbs, largest patient 643 lbs

The following topics, which have been the focus of extensive statistical analysis, compose the core of a successful medical weight loss program:

1. Patient motivation enhancement
2. Impact of time between office visits
3. Calorie intake optimization
4. Appetite control (suppression)
5. Computers in weight loss
6. Managing diseases affected by weight loss

APPETITE SUPPRESSION

The FDA removed fenfluramine from the market in September 1997. Prior to that this research project generated, and still has on file, 111,000 patient-days of fen-phen weight loss data. This allows statistical comparison of phen-fen results with current results. Results will be presented, documenting current weight loss up to 168% greater than phen-fen.

In addition, statistical data will be presented demonstrating optimal time between clinic visits and optimal caloric prescriptions in clinical weight loss.

COMPUTERS IN WEIGHT LOSS

The mathematics of weight loss is complex. Knowing if patients are on track to make goal weight is critical. Patients losing some weight, may not be positioned to make goal weight. The sooner these patients are repositioned, the higher the success rate. Computer performance printouts are extremely motivating. Attendees will get free access to the computer program.

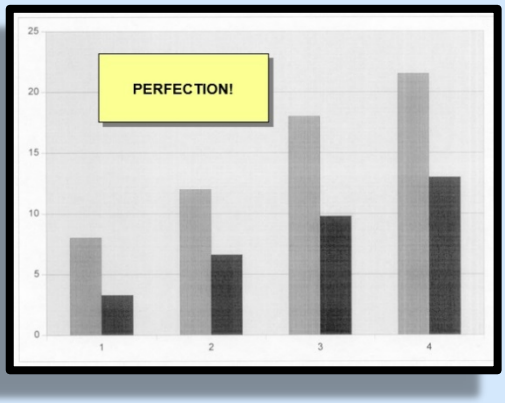


Figure 3: Black bars are expected weight (fat) loss. Gray bars are actual weight loss. Gray is greater than black due to water loss the first 2 or 3 weeks of treatment.

MANAGING DISEASE DURING WEIGHT LOSS

SEE THE LEFT COLUMN, YELLOW AREA PAGE 3: If you desire a practice where more drugs are stopped than started, effective weight loss is king. Get your type 2 diabetics off all insulin and drugs while maintaining normal blood sugars. Learn how to safely stop hypertensive meds during effective weight loss. Learn to manage toxins, hormones, and other fat-soluble substances in weight loss. Learn when to safely stop all prescribed medicines which are no longer needed secondary to effective weight loss.

The focus of this conference is not obesity or a specific disease, it is dedicated to serotonin, dopamine, norepinephrine, epinephrine, and thiol general optimization techniques.

Lecture Schedule and Speakers:

Friday April 27, 2018 (6.0 hours CME)

8:00 AM	Coffee and beverages
9:00 AM	Scientific foundation and history (Hinz)
10:30 AM	Break (1/2 hour)
11:00 AM	Reuptake inhibitors
12:30 PM	Lunch, provided one hour
1:30 PM	Parkinson, as a prototype dopamine disease
3:00 PM	Break (1/2 hour)
3:30 PM	Case studies
5:00 PM	Round table until no questions (no CME)

Saturday April 28, 2018 (6.0 hours CME)

8:00 AM	Coffee and beverages
9:00 AM	Dopamine disease management
10:30 AM	Break (1/2 hour)
11:00 AM	Wt. lose overview and approaches to appetite suppression
12:30 PM	Lunch, provided one hour
1:30 PM	Wt. loss – Motivation - Time between visits – Calorie prescription (CALRx) – Evaluating hunger
3:00 PM	Break (1/2 hour)
3:30 PM	Computers in bariatric medicine – managing secondary illness
5:00 PM	Round table until no questions (no CME)

Sunday April 29, 2018 (3.0 hours CME)

8:00 AM	Coffee and beverages
8:30 AM	Lab, Organic Cation Transporter-2 (OCT-2)
10:00 AM	Break (1/2 hour)
10:30 AM	Case study presentations
Noon	End of conference

Note: The registration fee covers one attendee and one guest (see the registrations fee section on page 7 [the next page]).

The focus of this conference is not obesity or a specific disease, it is dedicated to serotonin, dopamine, norepinephrine, epinephrine, and thiol general optimization techniques.

Marty Hinz, MD
Morgan Park Clinic Duluth, MN
President Clinical Research NeuroResearch Clinics

Steve Center, MD
BodyLogicMD of San Diego
San Diego, CA

Ross Stewart, PhD
Director Parkinson Clinics International
Dallas, TX

COURSE OBJECTIVES:

The participant will be able to explain the National Institute of Drug Abuse model as it relates to how reuptake inhibitors may deplete the centrally acting monoamines.

The participant will be able to discuss how each of the following may be induced under the National Institute of Drug Abuse reuptake inhibitor model.

1. Low efficacy
2. Placebo relapse
3. Suicidal ideation
4. Symptom relapse
5. Discontinuation syndrome
6. Drug tachyphylaxis in general

The participant will be able to describe strategies for effectively managing reuptake inhibitor-induced centrally acting monoamine depletion.

The participant will be able to determine optimal serotonin or catecholamine starting points in patient management.

The participant will be able to cite the impact of management decisions in response to inadequate serotonin and/or dopamine concentrations as the etiology of symptoms.

The participant will be able to describe the impact of competitive inhibition interaction between the centrally acting monoamines.

The participant will be able to describe the impact of conjugation interaction between centrally acting monoamines and thiols.

Course Objectives:

The participant will be able to cite why the drug L-dopa is the most effective Parkinson's disease treatment.

The participant will be able to describe carbidopa's mechanism of action and its potential impact on:

1. Worsening Parkinson's symptoms
2. Parkinson's death rate
3. L-dopa tachyphylaxis
4. Depletion of other systems
5. Dyskinesias and choreiform movement

The participant will be able to describe the published mechanism of action for Parkinson's on/off effect.

The participant will be able to describe functional status determination of the Organic Cation Transporters Type-2

The participant will be able to understand the process used to determine optimal appetite suppression efficacy in medical weight loss.

The participant will be able to cite optimal positioning for each of the following in medical weight loss.

1. Patient motivation enhancement
2. Impact of time between office visits
3. Calorie intake optimization
4. Appetite control (suppression)
5. Computers in weight loss
6. Managing diseases affected by weight loss

15 Hours AMA Category 1 CME

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The original research course

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Registration Fee	Before March 1	After Feb. 28
Regular Rate	$399	$599
Previously Attended a NeuroResearch Conference	$199	$399
Pay at the door: $699 for all		
Note: The registration fee covers the attendee and one guest. Medical education credit for the guest will cost an additional $200.		

For more information or to register, call NeuroResearch at 877-626-2220 **www.NeuroSupport.com**

INTENDED AUDIENCE: Physicians, psychiatrists, clinical psychologists, gastroenterologists, naturopaths, nurse practitioners, physician assistants and other healthcare providers licensed to manage patient problems relating to serotonin, dopamine, norepinephrine, epinephrine and/or thiols.

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Physician Assistants: NCCPA accepts *AMA PRA Category 1 Credit™* from organizations accredited by ACCME.

Commercial Support: Full notification of commercial support will be provided in the final program.

Criteria for Success: Statements of credit will be awarded based on the participant's attendance and submission of the activity evaluation form. A statement of credit will be available upon completion of an online evaluation/claimed credit form at http://akhcme.com/akhcme/pages/neuroresearch. You must participate in the entire activity to receive credit. If you have questions about this CME activity, please contact AKH Inc. at tbrignoni@akhcme.com.

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