

**23rd Annual International Symposium
on
Man and His Environment in Health and Disease**

**Special Focus
The Autonomic Nervous System and Its Relationship to Environmental
Pollutants Including the Cardiovascular System and Electromagnetic
Sensitivity**

**Sponsored by
American Environmental Health Foundation
and
University of North Texas Health Science Center**

Physician Accreditation/Credit:

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education and the American Environmental Health Foundation. The University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education is accredited by the ACCME to provide continuing medical education for physicians.

The University of North Texas Health Science Center at Fort Worth is accredited by the American Osteopathic Association to award continuing medical education to physicians.

The University of North Texas Health Science Center at Fort Worth designates this educational activity for a maximum of 24 Category 1 credits toward the AMA Physician's Recognition Award. Each physician should claim only those credits that he/she actually spent in the activity.

The University of North Texas Health Science Center anticipates this program for 24 hours in Category 2A CME credit hours, pending approval from the American Osteopathic Association.

Nursing Accreditation/Credit:

University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education, Provider #02588A, is approved provider of continuing nursing education by the Texas Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation. This activity

meets Type 1 criteria for mandatory continuing education requirements toward relicensure as established by the Board of Nurse Examiners for the State of Texas. This activity is approved for 28.8 Contact Hours.

To receive a certificate of successful completion, participants must attend the activity in its entirety and complete and return the activity evaluation credit request form.

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FINANCIAL CONSIDERATION

AEHF is a nonprofit organization that was founded in 1975 to provide education and research into Environmental Medicine. This year's Symposium is our 23rd Annual International Symposium and is our major vehicle for educating the medical professional.

Funding for the symposium is provided by registration fees from physicians and exhibitors. Proceeds from the AEHF store cover the shortfall between registration fees and expenses for the conference. AEHF does not receive grants or any outside financial support for our education. Donations are accepted and used toward research into environmental medicine.

INTRODUCTION

SYMPOSIUM PURPOSE

Since 1981, the International Symposium has been recognized as one of the most advanced medical forums in the world addressing the research and treatment of environmental effects on health and disease. The 2005 conference will focus on "The Autonomic Nervous System and Its Relationship to Environmental Pollutants Including the Cardiovascular System and Electromagnetic Sensitivity". This Conference presents the most current information available while providing guidelines to identify, diagnose, treat and to prevent environmentally triggered responses in the body.

GOALS OF THE MEETING

- ! To provide new insights in *The Autonomic Nervous System and Its Relationship to Environmental Pollutants* and the environmental causes behind many problems you see.
- ! To present new diagnostic and treatment modalities to help you improve the quality of care for your complex patients.
- ! To provide concepts, tools that will enhance your practice.

OBJECTIVES OF THE MEETING

- ! Improve the outcome of treating patients with sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Use new concepts and treatments to help better diagnose and manage many patients with environmentally triggered problems and sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Apply the concepts of this conference to your practice by using nutrition and environmental manipulation for the treatment of sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Use the information presented to enhance the effectiveness, cost-efficiency, and competitiveness of the

physician in relation to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.

INTENDED AUDIENCE

M.D.=s, D.O.=s, medical students, nurses, nutritionists and other health professionals interested in the concepts and practice of Environmental Medicine, Occupational Medicine and Toxicology.

EDUCATIONAL FORMATS

- ✂ Plenary
- ✂ Panels Discussions
- ✂ Case Studies
- ✂ Question & Answer Sessions.

CONFERENCE FORMAT

The AEHF Committee has selected some of the leading experts in the fields of chronic disease, nutrition and chemical sensitivity.

Each speaker=s presentation will last approximately 20 minutes and will be followed by a 10 minute question and answer session. All speakers are encouraged to use any and all appropriate audio/visual aids. (A brief outline of the speech is included in this booklet.)

GIVEN IN COOPERATION

William J. Rea, M.D., F.A.C.S.

Symposium Chairman,
American Environmental Health Foundation,
Environmental Health Center - Dallas,
Dallas, Texas

Bertie B. Griffiths, Ph.D.,

Environmental Health Center - Dallas
Dallas, Texas

Kaye H. Kilburn, M. D.

University of Southern California Medical Center
Keck School of Medicine
Los Angeles, CA

William J. Meggs, M.D., Ph.D.

Brody School of Medicine, East Carolina University
Department of Emergency Medicine
Greenville, NC

23rd ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT

Thursday, June 9, 2005

7:00-9:00 a.m. REGISTRATION

9:00 WELCOME/MODERATOR: William J. Rea, M.D., Doug Seba, Ph.D.

9:10 "Environmental Update 2005: Chemicals, Nerves and Electromagnetism", Doug Seba, Ph.D.

9:30 Q & A

9:40 "Dysautonomias and Dysrhythmias", Amer Suleman, M.D.

10:00 Q & A

10:10 MORNING BREAK

10:30 "ANS Assessment as an Indicator of Environmental Pollution Impacts on the Human Body",
Alexander Riftine, Ph.D.

10:50 Q & A

11:00 "Autonomic Nervous System in Environmentally Damaged Chronic Degenerative Disease and Hypersensitive
Patients", William J. Rea, M.D., FACS

11:20 Q & A

11:30 "Autonomic Innervation of Gut Mucosa-Associated Lymphoid Tissue", Javier Santos, M.D.

11:50 Q & A

12:00 LUNCH IN THE PANORAMA ROOM

1:30 MODERATOR: William J. Meggs, M.D., Ph.D.

"POTS - Autonomic Dysregulation and Mineral Deficiencies", Kalpana Patel, M.D.

1:50 Q & A

2:00 "RR Interval; Postural Hypotension - Shy-Drager", Kaye H. Kilburn, M.D.

2:20 Q & A

2:30 "Tilt Testing and Syncope: A Clinical Roadmap of the Autonomic Nervous System",
Freddy Abi-Samra, M.D.

2:50 Q & A

3:00 AFTERNOON BREAK

3:30 “Autonomic Dysfunction in Multiple Chemical Sensitivity”, Roy Fox, M.D.
3:50 Q & A

4:00 “A Pathophysiologic Approach to Treatment of Vasodepressor Syncope”, Freddy Abi-Samra, M.D.
4:20 Q & A

4:30 “Chronic Otitis Media with Effusion - The Allergy Connection”, David Hurst, M.D., Ph.D.
4:50 Q & A

5:00 PANEL DISCUSSION/CASE STUDIES: Wallace Rubin M.D.
“Autonomic Nervous System Environmental Pollutants and the Inner Ear”

6:00 ADJOURN

THURSDAY, JUNE 9, 2005

ABSTRACTS

AND

HANDOUTS

Objectives & Notes

Doug Seba, Ph.D.

Date of talk: Thursday, June 9, 2005,
9:10am.

P.O. Box 1417, #323
Alexandria, VA 22313

Phone: 703/949-1055

Training:

Current Job Description:	Independent Marine Scientist
Medical School/ University Attended	University of Miami, Coral Gables, Florida
Other Information:	40 years experience with chemicals and the environment.

SPEECH TITLE: “Environmental Update 2005: Chemicals, Nerves and Electromagnetism”

At the end of this Presentation, the participant should be able to:

1. To understand the connection between environmental stressors, particularly electromagnetic and xenobiotic chemicals, and adverse neurological processes.
2. To realize that such adverse health effects can occur at great distances in time and place from their environmental origin making diagnosis and treatment difficult.
3. References drawn from a mix of media, websites, and scientific publications relevant to the current timeline.

**ENVIRONMENTAL UPDATE 2005: CHEMICALS, NERVES AND
ELECTROMAGNETISM**

Douglas B. Seba

Dysfunction of the autonomic nervous system is quintessentially the definition of the typical environmental illness patient. Simply put, in almost all cases of environmental

illness, whether from chemical, physical, or biological sources, or a combination of them, nervous system symptoms are a keystone in both diagnosis and treatment of the patient. This Conference rightly acknowledges this relationship and draws focus to environmental pollutants, a theme of 23 years, because it is a root cause of environmental illness and unfortunately, pollution endures. In particular, this Conference will examine the effects of environmental pollutants on the neurological processes of the cardiovascular system, reflecting a principle interest of the AEHF, with special emphasis on one of the most insidious physical pollutants, electromagnetism.

Often called electronic smog in contemporary literature, mankind is being exposed to far more electromagnetic pollution than in any time in our planet's history. Like chemicals, electromagnetism appears to have two sides. On one hand, a study shows a two-fold risk increase of acoustic neuromas from cell phone use, while another study shows repetitive transcranial magnetic stimulation to improve mental focus. Both studies demonstrate that electromagnetism exposure has health effects. Just like chemicals and biologicals which have become pervasive in our environment, and often have adverse effects at great distances in time and location from their origins, so too, do electromagnetics. Outside of living in a Faraday cage, a patient and his or her heart are exposed to electromagnetic smog from things as close as their toaster and as distant as satellites or sun spots.

This is a general review to set the tone for the Conference. Highly selected examples will be drawn from a mix of media, website, and scientific publications relevant to the current timeline. Some examples of electromagnetic processes will be personally applied by the reviewer to ongoing research in wildlife anomalies in the Bitterroot mountains of Montana and the incursion of African dust storms into the Western hemisphere.

Objectives & Notes

Amer Suleman, M.D.

Date of talk: Thursday, June 9, 2005, 9:40am

The Heartbeat Clinic

Phone: 214/504-9942

4510 Medical Center Drive, Suite 208

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McKinney, TX 75069

Email:

asuleman@theheartbeatclinic.com

Training:

Current Job Description:	Private Practice in Electrophysiology
Medical School/ University Attended	King Edward Medical College
Internship:	SUNY at Buffalo
Residency:	SUNY at Buffalo
Board Certifications:	Electrophysiology; Cardiology; Internal Medicine

SPEECH TITLE: “Dysautonomias and Dysrhythmias”

At the end of this Presentation, the participant should be able to:

1. Understand interaction of autonomic nervous system with arrhythmias.
2. Understand Physiology of autonomic nervous system.
3. Understand role of management options for common arrhythmias.

The above information was provided by the Speaker.

Objectives & Notes

William J. Rea, M.D., FACS	Date of talk: Thursday, June 9, 2005, 11:00am
Environmental Health Center - Dallas 8345 Walnut Hill Lane, Ste. 220 Dallas, TX 75231	Phone: 214/368-4132 Fax: 214/691-8432 Email: wjr@ehcd.com
Training:	
Current Job Description:	M.D., President – Environmental Health Center – Dallas
Current Faculty Appointments:	Professor of Medicine, Capital University of Integrative Medicine, Washington, D.C.
Medical School/ University Attended	Ohio State University College of Medicine, Columbus, OH
Internship:	Parkland Memorial Hospital, Dallas, TX
Residency:	University of Texas SW Medical School, Dallas, TX
Board Certifications:	American Board of Surgery; American Board of Thoracic Surgery; American Board of Environmental Medicine
Other Information:	“Optimum Environments for Optimum Health and Creativity”

SPEECH TITLE: “Autonomic Nervous System Response in 100 Chemically Sensitive Patients”

At the end of this Presentation, the participant should be able to:

1. Be able to identify ANS dysfunction clinically
2. Be able to identify ANS dysfunction by laboratory
3. Be able to use this knowledge in their practice.

The above information was provided by the Speaker.

Objectives & Notes

Javier Santos, M.D.	Date of talk: Thursday, June 9, 2005, 11:30am
Hospital General Valle Hebron Institut Fundacio Recerca, Digestive Diseases Research Unit Paseo Valle Hebron 119-129 Barcelona, Barcelona 08035 Spain	Phone: 93/489-4035
	Fax: 93/489-4456
	Email: jsantos@vhebron.net
Training:	
Current Job Description:	Principal Investigator, Digestive Diseases Research Unit, Hospital Valle de Hebron, Barcelona, Spain
Medical School/ University Attended	University of Navarra, Spain
Internship:	Hospital Valle de Hebron, Barcelona, Spain
Residency:	Hospital Valle de Hebron, Barcelona, Spain
Board Certifications:	Not applicable

SPEECH TITLE: “Autonomic Innervation of Gut Mucosa-Associated Lymphoid Tissue”

At the end of this Presentation, the participant should be able to:

1. To understand that immune and nervous systems display a dynamic and interactive communication based upon the presence of common messengers and receptors.
2. This interaction is functionally relevant and may have important clinical implications.
3. Modulation of nerve-immune interactions in the gastrointestinal and respiratory tracts may influence the course of hypersensitivity and inflammatory disorders. Development of new drugs with blocking or enhancing activity against nerve-immune mediators and receptors may contribute to the prevention and treatment of these disorders.

The above information was provided by the Speaker.

Autonomic innervation of Gut-Mucosa-Associated Lymphoid tissue. Javier Santos, M.D., Ph.D. Hospital Universitario Valle de Hebron. Digestive System Research Unit. Barcelona, Spain.

Goals and Objectives

1. To understand that immune and nervous systems display a dynamic and interactive communication based upon the presence of common messengers and receptors.
2. This interaction is functionally relevant and may have important clinical implications.
3. Modulation of nerve-immune interactions in the gastrointestinal and respiratory tracts may influence the course of hypersensitivity and inflammatory disorders. Development of new drugs with blocking or enhancing activity against nerve-immune mediators and receptors may contribute to the prevention and treatment of these disorders.

Outline

The mucosal surfaces of the body are the first and critical location where immunogenic particles and molecules (food and microorganism-derived antigens) gain access to the immune system. Antigen processing in this region will determine whether an immunological balanced or unbalanced response (anaphylactic reaction) or a permissive one (tolerance) is mounted. Mucosal membranes are covered by epithelial layers. In the gastrointestinal and respiratory systems, classical effector cells of immune reactions (lymphocytes, eosinophils, mast cells, neutrophils, macrophages and dendritic cells) are normally present. Non-traditional immune cells, including epithelial, mesenchymal (fibroblasts, myofibroblasts, muscle cells), endothelial and nerve cells and also acellular components such as the extracellular matrix may also display potentially relevant effector and modulatory functions in antigen processing and immunologic responses. In addition, food itself may be also immunomodulatory.

Our knowledge of the immunoregulatory effects of nerves and neuropeptides has grown tremendously. Considerable and converging evidence has now established the existence of multidirectional communication between the neural and immune systems. This interaction involves most immune cells present in mucosal surfaces, and both the central and peripheral nervous systems, including efferent and afferent subdivisions of the autonomic nervous system, sympathetic and parasympathetic, as well as the enteric nervous system. Moreover, those studies have shown the functional relevance of neuro-

immune interactions in regulating immunological and inflammatory events in mucosal surfaces, by influencing the transport of macromolecules across the epithelial surface, the expression of adhesion molecules, the release of cytokines, chemokines, neurotransmitters, neuropeptides, and other regulatory molecules that participate in the trafficking and homing of immune cells in the mucosal layers, the growth and remodeling of nerves, and even the apoptotic cascade. A good clinical correlate are food-allergic reactions that may display a broad spectrum of clinical manifestations involving most tissues in the body, although it is generally accepted that primary events predominantly occur at the level of the gastrointestinal mucosa.

A distinct characteristic of the complex neuroendocrine and immune systems is the high level of integration of both systems which together provide the organism with an ultra-fine homeostatic balance. The neuroendocrine system modulates the function of the immune system through the release of neuropeptides, neurohormones and neurotransmitters. In addition, a primary or counteracting immunoregulatory role for immune cells has also been reported which comprises the effects of mediators released by immunocompetent cells on neuroendocrine function. It is now clear that immune cells are able to synthesize and release neuropeptides and even classical hormones such as growth hormone or prolactin, and that endocrine and neural cells can produce a broad array cytokines originally described as being part of the repertoire of immune cells. This conference will focus mainly on the efferent limb of the neuro-immune interactions, that is the effect of neural mediators on immune cells, particularly lymphocytes and mast cells. For this purpose anatomical and functional evidence for the presence and relevance of neural and neuropeptide-containing fibers in lymphoid tissues and immunocompetent cells will be reviewed. Finally, experimental and clinical data supporting the potential relevance of this interplay and its significance in the management of food allergy and other inflammatory disorders will be briefly exposed.

Conclusions

Central and peripheral nervous system manipulation by psychological (hypnosis, behavioral conditioning), pharmacological and other modalities of intervention could turn out to be as very valuable weapons against the deleterious effects of environmental stressors on the immune system. In particular, increasing awareness of the major role of gastrointestinal inflammation in the development of local and systemic adverse reactions to food, microbial and chemical components may open new avenues in the treatment and prevention of environmental-related inflammatory disorders.

References

- 1.Santos J, Bienenstock J, Perdue MH. Innervation of lymphoid tissue and functional consequences of neurotransmitter and neuropeptide release. In: Brostoff J, Challacombe SJ, eds. *Food Allergy and Intolerance*, 2nd ed. W.B. Saunders, London, 2002. 51-67.
- 2.Felten DL, Felten SY, Carlson SL, Olschowska JA, Livnat S. Innervation of lymphoid tissue. *J Immunol* 1985; 135: 755s-765s.
- 3.Madden KS, Felten DL. Experimental basis for neural-immune interactions. *Physiol Rev* 1996; 75: 77-106.
- 4.Straub RH & Besedovsky HO. Integrated evolutionary, immunological, and neuroendocrine framework for the pathogenesis of chronic disabling inflammatory diseases. *FASEB J* 2003; 17:2176–2183.
- 5.Elenkov I Chrousos GP. Stress Hormones, Proinflammatory and Antiinflammatory Cytokines, and Autoimmunity. *Ann NY Acad Sci.*2002; 966: 290-303.

Objectives & Notes

Kalpana Patel, M.D.	Date of talk: Thursday, June 9, 2005, 1:30pm
Allergy and Environmental Health Center - Buffalo 65 Wehrle Dr. Buffalo, NY 14225	Phone: 716/833-2213
	Fax: 716/833-2244
Training:	
Current Job Description:	President of EHC-Buffalo, President of American Board of Environmental Medicine
Current Faculty Appointments:	Asst. Professor of Pediatrics at SUNY Buffalo
Medical School/ University Attended	B.J. Medical School, India
Internship:	Pediatrics
Residency:	Pediatrics
Board Certifications:	Pediatrics/Environmental Medicine

SPEECH TITLE: “POTS - Autonomic Dysregulation and Mineral Deficiencies”

At the end of this Presentation, the participant should be able to:

1. Understand autonomic deregulation in postural orthostatic tachycardia syndrome and syncope.

The above information was provided by the Speaker.

Objectives & Notes

Kaye H. Kilburn, M.D.	Date of talk: Thursday, June 9, 2005, 2:00pm
University of Southern California Medical Center, Keck School of Medicine Bldg 7/7401 1000 So. Fremont St. Alhambra, CA 91803	Phone: 626/457-4202
	Fax: 626/457-4203
	Email: kilburn@usc.edu
Training:	
Current Job Description:	Ralph Edgington Professor of Internal Medicine, University of Southern California Keck School of Medicine
Medical School/ University Attended	University of Utah College of Medicine
Internship:	Western Reserve, University Hospitals Cleveland, Internal Medicine
Residency:	University of Utah Medicine, Pathology; Duke, Cardiopulmonary Physiology; University of London Cardiology
Board Certifications:	Diplomat American Board of Internal Medicine, American Board of Preventive Medicine Occupational Health
Other Information:	Books: <i>Chemical Brain Injury</i> and <i>Endangered Brains</i> <i>Mold and Mycotoxins</i> , (Kilburn, KH ed) Written several articles

SPEECH TITLE: “RR Interval; Postural Hypotension - Shy-Drager”

At the end of this Presentation, the participant should be able to:

1. Put the heart and vessels in perspective for effects of inhaled chemicals.
2. Understand measurements and interpretation heart rate monitoring.
3. What is and is not possible to learn from sinus arrhythmia.

The above information was provided by the Speaker.

**RR INTERVAL POSTURAL HYPOTENSION–SHY-DRAGER
IN ADULTS EXPOSED TO CHLORINE-CRESYLATE,
HYDROGEN SULFIDE,
MOLDS AND OTHER CHEMICALS
SIGNS OF AUTONOMIC NERVOUS SYSTEM
DYSFUNCTION?**

**Kaye H. Kilburn, M.D.
and
Bradford E. Hanscom**

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RR Interval – Postural Hypotension – Shy Drager Autonomic Failure

**Kaye H. Kilburn
Bradford E. Hanscom**

Background and Objective: Vagal heart variability can be monitored by recording beat to beat or RR intervals during deep slow breathing that exaggerates sinus arrhythmia, the normal RR variation with breathing. We determined whether a one day exposure to a chemical mixture of chlorine and cresylate affected RR variation 3 years after the incident. Normally heart rate increases during the inspiratory phase of respiration and it decreases in the expiratory phase. Vagus nerve regulated sinus arrhythmia is reduced or abolished by aging, diabetes mellitus and solvent exposure. The RR study was extended to 4 groups of people exposed to hydrogen sulfide, exposed to mold and mycotoxins, exposed to a variety of chemicals and to community control people.

Methods: We studied 58 adult subjects who had inhaled chlorine-cresylate 3 years earlier matched to 22 adult subjects unexposed to chemicals. A standard electrocardiograph machine was interfaced to an IBM compatible computer to record and time ECG-R waves during deep breathing at a rate of 6. An algorithm was developed to process the wave form variation so as to time all RR intervals and to create a confidence band of (\pm 10% mean time). The percentage of beats outside the confidence band were compared between exposed and control. Small R-waves when the chest was inflated and premature contractions were difficult to recognize. After the chlorine study a dedicated (2 lead and ground) electrocardiogram recorded R-waves with this patient lying after a 3-minute rest then they were guided to breathe 6 times per minute. I marked R-waves visually on 1 minute replays to calculate RR intervals and their means and standard deviations.

Results: Chlorine exposed subjects had 32.6% of beats outside this band compared to 47.7% of unexposed subjects ($p < 0.014$). The standard deviation of the RR intervals were 22.7 and 31.2 respectively ($p < 0.043$). Thus sinus arrhythmia was reduced after chlorine. For other groups mean Sd RR was 93 (range 12 to 253) in unexposed people, 93.3 in sewer gas exposed, 63.6 in hog lagoon exposed, and 96 in the chemical and mold group. Frequency distribution plots were asymmetrical with the median left of the mean in all groups including matched controls. Regression showed an age coefficient of -0.92 per year and a constant of 139.8. Only the hog lagoon exposed were different from community controls, but they were not different from their matched controls. No feature or range of RR was predicted by demographics or neurobehavioral test scores.

Conclusion: Chlorine exposure reduced the respiratory variation of the heart rate. This effect on vagal function paralleled adverse effects on balance, choice reaction time, vision: color discrimination and visual field performance. Being downwind of hog manure lagoons also reduced variability but otherwise hydrogen sulfide, mold and mycotoxins and neurotoxic chemicals did not affect heart rate control (sinus arrhythmia).

Second Approach: The failure to adjust to orthostatic stress is easily measured by contrasting standing with lying blood pressure.

In a series of patients there were several patterns of response for maintaining standing blood pressure. The most frequent response was an increase in heart rate, next was an increase in systolic blood pressure. Without either of these responses a postural shift to standing caused blood pressure to fall below the lying level although syncope or lightheadedness were not observed. The frequencies of these responses are compared in 3 groups of patients and in control subjects.

Objectives & Notes

Freddy Abi-Samra, M.D.	Date of talk: Thursday, June 9, 2005, 2:30pm
Ochsner Clinic 1514 Jefferson Hwy New Orleans, LA 70121	Phone: 504/842-4036
	Fax: 504/842-5823
	Email: fabisamra@ochsner.org
Training:	
Current Job Description:	Director Section of Electrophysiology, Department of Cardiology
Medical School/ University Attended	American University of Beirut School of Medicine
Internship:	American University of Beirut Medical Center
Residency:	American University of Beirut Medical Ctr., Dept. of Internal Medicine
Board Certifications:	American Board of Internal Medicine, American Board of Cardiology

SPEECH TITLE: “Tilt Testing and Syncope: A Clinical Roadmap of the Autonomic Nervous System”

At the end of this Presentation, the participant should be able to:

1. Understand and describe the schematics of autonomic control of the cardiovascular system.
2. Understand the value of tilt testing in stressing/evaluating the integrity of the various components of the autonomic nervous system.
3. Understand the difference between neurocardiogenic syncope and syncope do to autonomic dysfunction.

The above information was provided by the Speaker.

Objectives & Notes

Roy Fox, M.D.	Date of talk: Thursday, June 9, 2005, 3:30pm
Nova Scotia Environmental Health Centre P.O. Box 2130, 3064 Highway #2 Fall River, Nova Scotia B2T 1K6 Canada	Phone: 902/860-0551
	Fax: 902/860-2046
	Email: roy.fox@cdha.nshealth.ca
Training:	
Current Job Description:	Director, Nova Scotia Environmental Health Centre
Current Faculty Appointments:	Professor of Medicine; Dalhousie University; N.S., Canada
Medical School/ University Attended	Newcastle Upon Tyne, UK
Internship:	Newcastle Teaching Hospitals
Residency:	Royal Free Hospital, London, England
Board Certifications:	FRCPC (U.K. – Int. Medicine); FRCPC (Gastroenterology)
Other Information:	Master of Environmental Studies (Dalhousie) 2001 – Thesis “Multiple Chemical Sensitivity and the Environment”: “Env. Sensitivities in Dialysis Unit” Indoor

	Air 1999. 'Environment & MCS' Indoor Air 2002: "Adaptation in Individuals with Heightened Sensitivity to Combined Environmental Factors" Archives of Complex Env. Studies 2003.
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SPEECH TITLE: "Autonomic Dysfunction in Multiple Chemical Sensitivity"

At the end of this Presentation, the participant should be able to:

1. The symptoms & signs of autonomic dysfunction in MCS.
2. Research results which demonstrate dysfunction.
3. Overlap with Fibromyalgia & chronic fatigue syndrome and MCS.

The above information was provided by the Speaker.

**AUTONOMIC DYSFUNCTION IN MULTIPLE CHEMICAL SENSITIVITY
(MCS)**

Roy Fox ¹, Barbara Adams ¹, Tara Sampalli ¹, Michel Joffres ^{1,2}

¹ Nova Scotia Environmental Health Centre, Fall River, Nova Scotia, Canada

² Community Health and Epidemiology, Faculty of Medicine, Dalhousie University,
Halifax, Canada

Multiple chemical sensitivity (MCS) is a chronic condition which overlaps with other chronic illnesses, namely chronic fatigue syndrome and Fibromyalgia. In our patient population who fulfill the published consensus criteria for MCS, almost half the patients also fulfill the criteria for chronic fatigue syndrome (Canadian consensus criteria 2003) and Fibromyalgia (American College of Rheumatology) or both. Autonomic dysfunction has been reported in both of these two conditions. The symptoms of patients with MCS indicate a high likelihood of autonomic dysfunction. In an earlier study of 351 patients with MCS, 75% reported light-headedness, 78% reported cold hands or feet, 62% experienced racing heart not related to exercise or emotion, 18% reported coloured finger tips and about 36% white painful fingers in cold as symptoms experienced since the onset

of their illness. About 50% reported light-headedness and 42% reported cold hands or feet in the intense range.

In a pilot study of 7 women with MCS, age matched with 7 healthy women, it was found that the autonomic response to exercise (6 minute walk on treadmill at self-selected walk speed) was significantly different. For patients with MCS the heart rate change was lower at 6 minutes, and the mean percentage change in diastolic blood pressure was higher at 3 minutes in comparison to control subjects. At the same time as these changes were noted the perceived exertion was higher for the MCS patients.

In a pilot study of challenge booth testing of individuals with and without MCS, a clear difference existed in the adaptation to the baseline experimental protocols. In the patient group, 25% required up to 4 sessions to adapt while at least 50% required at least 2 sessions. All the controls except one required only a single session to adapt to the baseline protocols. Cold feet and hands, fatigue, and numbness in fingers and toes were reported in the study as some of the symptoms experienced by patients when exposed to glue and dryer sheet.

The main function of the ANS is to maintain homeostasis in the face of changing demands. It is proposed that repetitive environmental stress requires repetitive adaptation and ultimately through damage or exhaustion, impaired function results. ANS dysfunction and impaired homeostasis accounts for many of the symptoms of patients with MCS. Patients are complex and there are other factors. A model of how ANS dysfunction fits in the spectrum of symptoms is presented.

Objectives & Notes

Freddy Abi-Samra, M.D.	Date of talk: Thursday, June 9, 2005, 4:00pm
Ochsner Clinic	Phone: 504/842-4036
1514 Jefferson Hwy	Fax: 504/842-5823
New Orleans, LA 70121	Email: fabisamra@ochsner.org

Training:	
Current Job Description:	Director Section of Electrophysiology, Department of Cardiology
Medical School/University Attended:	American University of Beirut School of Medicine
Internship:	American University of Beirut Medical Center
Residency:	American University of Beirut Medical Ctr., Dept. of Internal Medicine
Board Certifications:	American Board of Internal Medicine, American Board of Cardiology

SPEECH TITLE: “A Pathophysiologic Approach to the Treatment of Vasodepressor Syncope”

At the end of this Presentation, the participant should be able to:

1. Understand the pathophysiology of vasodepressor syncope.
2. Have adequate appreciation of the value of correct therapeutic schemes of VDS.
3. Tailor treatment of VDS based on provoked pathophysiologic disturbances of the autonomic cardiovascular regulation.

The above information was provided by the Speaker.

Objectives & Notes

David Hurst, M.D., Ph.D.	Date of talk: Thursday, June 9, 2005, 4:30pm
23 Spring St., Suite D Gorham, ME 04074	Phone: 207/883-6464
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Training:	
Current Job Description:	Private Practice – Otolaryngic Allergy
Current Faculty Appointments:	Tufts University, Boston, Massachusetts
Medical School/ University Attended	Indiana University
Internship:	Portland, ME
Residency:	Tufts – B.U. – Boston, Massachusetts
Board Certifications:	AAO-UNS, AAOA
Other Information:	Ph.D. – Uppgala, Sweden

SPEECH TITLE: “Chronic Otitis Media with Effusion - The Allergy Connection”

At the end of this Presentation, the participant should be able to:

1. Understand how acute and chronic otitis media differ.
2. Understand how the middle ear behaves as a target organ of allergic inflammation.
3. Understand how allergies account for the risk factors and development of OME.

The above information was provided by the Speaker.

Chronic Otitis Media with Effusion – The Allergy Connection

David Hurst, M.D., Ph.D.
Portland, ME

Abstract:

Methods to control chronic OME, otorhea, and draining mastoid cavities will continue to fail as long as the effects of allergy on middle ear mucosa are ignored. We have demonstrated that all cells essential to a Th-2 driven immune response are active in the mucosa in a majority of ears from atopics with chronic OME. We will review studies which prove that the middle ear serves as target organs of allergy, present studies which prove allergy management to be > 83% efficacious, and demonstrate why chronic otitis media with effusion is an allergic disease.

References:

(PMID = Pub Med ID)

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PMID: 11551246
more info and handouts:

David Hurst, MD, PhD 207-883-6464

<http://www.earallergy.com> - go to Course Materials and Handout section at bottom of selection panel.

Objectives & Notes

Wallace Rubin, M.D.	Date of talk: Thursday, June 9, 2005, 5:00pm
3434 Houma Boulevard Metairie, LA 70006-4226	Phone: 504/888-8800
	Fax: 504/455-6796
	Email: wrubinmd@bellsouth.net

Training:	
Current Job Description:	Solo Practioner
Current Faculty Appointments:	L.S.U. – Prof.
Medical School/ University Attended	University of Illinois
Internship:	Cook County Hospital
Residency:	Tulane University
Board Certifications:	Otolaryngology
Other Information:	Dizziness – Etiologic Approach to Management

CASE STUDY: “Autonomic Nervous System Environmental Pollutants and the Inner Ear”

At the end of this Presentation, the participant should be able to:

1. Evaluate inner ear function
2. Determine mechanisms causing abnormality
3. Treatment options

The above information was provided by the Speaker.

ENVIRONMENTAL TRIGGERS LEAD TO ABNORMAL RESPONSES FOR THE
RESPIRATORY SYSTEM
(EAR, NOSE, THROAT, LARYNX, AND LUNGS)

Symptoms of chemical sensitivity typically are multiple in nature. Usually, one main organ is affected with secondary symptoms occurring in others. End-organ responses are often in the smooth muscles of the cardiovascular, gastrointestinal, urogenital, respiratory systems, or in the nervous system. Also, common early responses may occur in the skin (such as nonjaundice yellowing or edema) or other body organs.

At their onset, symptoms of chemical sensitivity are almost always reversible. As end-organ involvement increases, however, responses are more difficult to decipher and reverse.

Pollutant damage can occur at the main site of pollutant entry or in the detoxifying organ or it can be random, affecting any end-organ. Usually, however, the weakest end-organ, that which has been genetically damaged or previously harmed by trauma or exposure, is the first affected.

The sicker the patient with chemical sensitivity, the more diverse and multiple are his responses to a large number of individual incitants, suggesting primary and secondary organ involvement. For example, a patient develops rhinitis on exposure to formaldehyde. Later in the course of his illness, symptoms and signs of cystitis and colitis develop in addition to the rhinitis.

**23rd Annual International Symposium
on
Man and His Environment in Health and Disease**

**Special Focus
The Autonomic Nervous System and Its Relationship to Environmental
Pollutants Including the Cardiovascular System and Electromagnetic
Sensitivity**

**Sponsored by
American Environmental Health Foundation
and
University of North Texas Health Science Center**

Physician Accreditation/Credit:

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education and the American Environmental Health Foundation. The University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education is accredited by the ACCME to provide continuing medical education for physicians.

The University of North Texas Health Science Center at Fort Worth is accredited by the American Osteopathic Association to award continuing medical education to physicians.

The University of North Texas Health Science Center at Fort Worth designates this educational activity for a maximum of 24 Category 1 credits toward the AMA Physician's Recognition Award. Each physician should claim only those credits that he/she actually spent in the activity.

The University of North Texas Health Science Center anticipates this program for 24 hours in Category 2A CME credit hours, pending approval from the American Osteopathic Association.

Nursing Accreditation/Credit:

University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education, Provider #02588A, is approved provider of continuing nursing education by the Texas Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation. This activity

meets Type 1 criteria for mandatory continuing education requirements toward relicensure as established by the Board of Nurse Examiners for the State of Texas. This activity is approved for 28.8 Contact Hours.

To receive a certificate of successful completion, participants must attend the activity in its entirety and complete and return the activity evaluation credit request form.

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FINANCIAL CONSIDERATION

AEHF is a nonprofit organization that was founded in 1975 to provide education and research into Environmental Medicine. This year's Symposium is our 23rd Annual International Symposium and is our major vehicle for educating the medical professional.

Funding for the symposium is provided by registration fees from physicians and exhibitors. Proceeds from the AEHF store cover the shortfall between registration fees and expenses for the conference. AEHF does not receive grants or any outside financial support for our education. Donations are accepted and used toward research into environmental medicine.

INTRODUCTION

SYMPOSIUM PURPOSE

Since 1981, the International Symposium has been recognized as one of the most advanced medical forums in the world addressing the research and treatment of environmental effects on health and disease. The 2005 conference will focus on “The Autonomic Nervous System and Its Relationship to Environmental Pollutants Including the Cardiovascular System and Electromagnetic Sensitivity”. This Conference presents the most current information available while providing guidelines to identify, diagnose, treat and to prevent environmentally triggered responses in the body.

GOALS OF THE MEETING

- ! To provide new insights in *The Autonomic Nervous System and Its Relationship to Environmental Pollutants* and the environmental causes behind many problems you see.
- ! To present new diagnostic and treatment modalities to help you improve the quality of care for your complex patients.
- ! To provide concepts, tools that will enhance your practice.

OBJECTIVES OF THE MEETING

- ! Improve the outcome of treating patients with sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Use new concepts and treatments to help better diagnose and manage many patients with environmentally triggered problems and sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Apply the concepts of this conference to your practice by using nutrition and environmental manipulation for the treatment of sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Use the information presented to enhance the effectiveness, cost-efficiency, and competitiveness of the physician in relation to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.

INTENDED AUDIENCE

M.D.=s, D.O.=s, medical students, nurses, nutritionists and other health professionals interested in the concepts and practice of Environmental Medicine, Occupational Medicine and Toxicology.

EDUCATIONAL FORMATS

- ✂ Plenary
- ✂ Panels Discussions
- ✂ Case Studies

CONFERENCE FORMAT

The AEHF Committee has selected some of the leading experts in the fields of chronic disease, nutrition and chemical sensitivity.

Each speaker's presentation will last approximately 20 minutes and will be followed by a 10 minute question and answer session. All speakers are encouraged to use any and all appropriate audio/visual aids. (A brief outline of the speech is included in this booklet.)

GIVEN IN COOPERATION

William J. Rea, M.D., F.A.C.S.

Symposium Chairman,
American Environmental Health Foundation,
Environmental Health Center - Dallas,
Dallas, Texas

Bertie B. Griffiths, Ph.D.,

Environmental Health Center - Dallas
Dallas, Texas

Kaye H. Kilburn, M. D.

University of Southern California Medical Center
Keck School of Medicine
Los Angeles, CA

William J. Meggs, M.D., Ph.D.

Brody School of Medicine, East Carolina University
Department of Emergency Medicine
Greenville, NC

23rd Annual International Symposium on Man and His Environment

Schedule

Friday, June 10, 2005

8:30 ANNOUNCEMENTS/MODERATOR: Kaye H. Kilburn, M.D.

9:05 "Neurogenic Inflammation, Autonomic Dysfunction, and Chemical Sensitivity", William J. Meggs, M.D., Ph.D.

9:25 Q & A

9:35 "Electromagnetic Sensitivity and the ANS", Cyril Smith, Ph.D.

9:55 Q & A

10:05 MORNING BREAK WITH EXHIBITORS

10:30 "The Synergy of Mind and Heart", Martha Stark, M.D.

10:50 Q & A

11:00 "Immune Mechanism Associated with Cows Milk Intolerance", Colin Little M.D.

11:20 Q & A

11:30 "Master Chemicals Suppressing Multiple Chemical Intolerances", Robert W. Gardner, Ph.D.

11:50 Q & A

12:00n OPEN LUNCH

MODERATOR: Jean Monro, M.D.

1:30 "Autonomic Epiphenomena of Segmental Radiculo - Neuropathy", Steven Goodman, M.D.

1:50 Q & A

2:00 "Toxicity of Autonomic Ganglia (and Plexi) Diagnosis & Treatment", Dietrich K. Klinghardt, M.D., Ph.D.

2:20 Q & A

2:30 "Polybrominated Diphenyl Ether and Human Health", John Laseter, Ph.D.

2:50 Q & A

3:00 AFTERNOON BREAK WITH EXHIBITORS

3:30 "Autonomic Disorders with Breast Implant Reconstruction", Lucie Lessard, M.D.

3:50 Q & A

4:00 “Intramuscular Stimulation Therapy for Autonomic Epiphenomena of Segmental Radiculo-Neurotherapy”,
Steven Goodman, M.D.

4:20 Q & A

4:30 “Chemical-Induced Developmental Neurotoxicity”, Mohamed B. Abou-Donia, Ph.D.

4:50 Q & A

5:00 PANEL DISCUSSION/CASE STUDIES: Allan Lieberman M.D.

“The Dysautonomic Pattern of Environmentally Triggered Disease”

6:00 RECEPTION WITH EXHIBITORS

FRIDAY, JUNE 10, 2005

ABSTRACTS

AND

HANDOUTS

Objectives & Notes

William J. Meggs, M.D., Ph.D.

Date of talk: Friday, June 10, 2005, 9:05am

Brody School of Medicine, East
Carolina University
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Phone: 252/744-2954
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Training:

Current Job Description:	Medical School Professor
Current Faculty Appointments:	Allergy & Immunology Brody School of Medicine
Medical School/ University Attended	University of Miami School of Medicine
Internship:	University of Rochester
Residency:	University of Rochester; Fellowships: NIH, NYU
Board Certifications:	Medical Toxicology, Internal Medicine, Emergency Medicine
Other Information:	Co-author of "The Inflammation Cure", Scientific Progress in Understanding Gulf War Illnesses.

SPEECH TITLE: "Neurogenic Inflammation, Autonomic Dysfunction, and Chemical Sensitivity"

At the end of this Presentation, the participant should be able to:

1. To know the role of neurogenic inflammation in chemical sensitivity.
2. To know the types of autonomic reactions to chemical exposures.
3. To know how neurogenic inflammation may mediate autonomic reactions to chemical exposures.

The above information was provided by the Speaker.

Neurogenic Inflammation, Autonomic Dysfunction, and Chemical Sensitivity

William Joel Meggs, MD, PhD
Brody School of Medicine
East Carolina University
Greenville, NC
USA

Chemical sensitivity is individual susceptibility to environmental chemicals at levels generally considered safe. Patients with such susceptibilities can have associated autonomic dysfunction. An early report of this came from Arthur Coca, who reported tachycardia in association with exposures. Recent studies have established an important role of neurogenic inflammation in chemical sensitivity. These studies will be reviewed. The reconciliation of autonomic dysfunction with neurogenic inflammation involves an understanding of the role of neurogenic inflammation in host defense and the role of the central nervous system in regulating neurogenic inflammation. Autonomic dysfunction may arise from abnormalities in the regulation of autonomic functions.

Objectives & Notes

Cyril Smith, Ph.D.

Date of talk: Friday, June 10, 2005, 9:35am

36 Westminster Road
Ellesmere Park
Eccles, Manchester M30-9EA
U.K.

Phone: 011/44-161-789-4768
Email: cyril.smith@which.net

Training:

Current Job Description:	Retired
Medical School/ University Attended	Imperial College, University of London, England
Other Information:	Smith C.W. (2002) <i>Effects of Electromagnetic Fields in the Living Environment</i> . Proc. Intl. Conf. Electromagnetic Environments and Health in Buildings, Royal College of Physicians, London, 16-17 May, 2002. In: Clements-

Croome D (Ed.). *Electromagnetic Environments and Health in Buildings*. London: Taylor & Francis, October 2003. Chap. 3, pp. 53-118. ISBN 0-415-316-561
Smith CW. (2003) Guest Editorial – Straws in the Wind. *Journal Alternative and Complementary Medicine* **9(1)**: 1-6.
Smith CW. (2003) Guest Editorial – Energy Medicine United. *Complementary Therapies in Nursing and Midwifery* **9(4)**:169-175 (Nov 2003).
Smith CW. (2004) Quanta and Coherence Effects in Water and Living Systems. *J Altern Complement Med.* 10(1);69-78.

SPEECH TITLE: **“Electromagnetic Sensitivity and the ANS”**

At the end of this Presentation, the participant should be able to:

1. Recognize electromagnetic sensitivity in patients
2. Appreciate the importance of endogenous and exogenous frequencies
3. Understand the relation between frequency and the ANS

The above information was provided by the Speaker.

**23rd ANNUAL INTERNATIONAL SYMPOSIUM
ON MAN & HIS ENVIRONMENT**

Abstract

**“Electromagnetic Sensitivity and the ANS”
and
“ANS Involvement in Chemical and Electromagnetic Sensitivities”
Cyril Smith, Ph.D.**

The first part of these two presentations is intended to enable the participant to recognize Electromagnetic Sensitivities in patients, to appreciate the importance of endogenous and exogenous frequencies and the relationship between these frequencies and the autonomic nervous system.

The second part is intended to explain the duality between frequencies and chemical structure and their relation to autonomic nervous system stability criteria and interactions in living systems. The effects of interactions between chemical in the body and environmental electromagnetic fields is explained in terms of the endogenous frequencies on acupuncture meridians and linkages between specific acupuncture points and points in the autonomic nervous system.

The conclusions are that Voll's link between these specific acupuncture points and the ANS gives an insight into how environmental frequencies can interact with the ANS. The Multiple Frequency Effect in coherent systems shows how the frequency signatures of chemicals can behave like environmental frequencies and vice versa.

References to the writer's publications on “Electrical Hypersensitivity and Water Phenomena”, and previous presentations at these International Annual Symposia on “Man and His Environment in Health and Disease” held in Dallas, Texas, from 1986-2000 are listed in the Handout which also includes some definitions of electromagnetic quantities.

A set of “Notes for Patients on Electrical Sensitivities” written for the Breakspear hospital, England are also appended as these may be found useful for clinicians to have available.

23rd ANNUAL INTERNATIONAL SYMPOSIUM ON MAN & HIS ENVIRONMENT

Handout for Presentations

“Electromagnetic Sensitivity and the ANS” and “ANS Involvement in Chemical and Electromagnetic Sensitivities”

Cyril Smith, Ph.D.

Retired from The University of Salford, M5 4WT, UK.

1. Background

Since 1974 – The writer has been involved since 1974 in research on the ‘Interactions of Electromagnetic Fields with Bio-Materials and Living Systems’. He cooperated in this with Professor Herbert Fröhlich FRS. An early conclusion of this work was that there were anomalous magnetic field effects in water and living biological systems and that these were only explicable in terms of coherence phenomena giving long-range order.

Since 1982 The writer first became involved in the diagnosis and therapy of patients ‘Hypersensitive to their Electromagnetic Environment’ in 1982 at the request of Dr. Jean Monro. Work with her electrically hypersensitive patients and with those of Dr. W.J. Rea has given an insight into the extremes of sensitivity of which living systems are capable as evidenced when their ANS control mechanisms fail.

The writer’s publications on “Electrical Hypersensitivity and Water Phenomena” and some definitions of electromagnetic quantities are listed at the end of this document. A set of “Notes for Patients on Electrical Sensitivities” are also appended as these may be found useful for clinicians to have available.

2. Electromagnetic Hypersensitivity

Electromagnetically sensitive patients almost invariably have a history of hypersensitivities to many chemicals, and/or foods and particulates. The autonomic nervous system appears to be the first body system to become involved. Patients may react within seconds to something in their environment. They can readily distinguish *verum* from *placebo*. The frequency and its coherence seemed to be the clinically important parameter. There is a threshold for the intensity or amplitude of the field at the patient for the onset of any effects but, once this is exceeded its value usually matters little until the onset of thermal effects; it is the frequency which is important.

The clinical effects of frequencies are unique to each individual. Some frequencies are stimulatory or therapeutic and these usually alternate with depressive or stressful frequencies. This alternation of the stimulatory-depressive effect of frequencies is a general phenomenon with few exceptions. It resembles the effects produced by the serial dilution of an allergen; higher frequencies resemble higher dilutions or potencies.

The clinically effective frequencies range from near circadian (0.4 milliHertz, 2,500 sec/cycle or, 42 min/cycle) to above microwave frequencies (1 GigaHertz = 10^9 Hz) and sometimes to optical frequencies for patients hypersensitive to sunlight.

Identical reactions can be triggered in a patient by chemical means and neutralized with electrical frequencies or triggered electrically and neutralized chemically. The clinical effects of environmental frequencies or chemicals can be reproduced by water contained in sealed glass ampoules after its exposure to coherent frequencies of an alternating magnetic field *without any chemical contact*. The unexposed water produces no clinical effects.

Chemical toxicity in these patients is manifest through the appearance of chemicals' *frequency signatures (frequencies arising from H-bonding between water and the chemical)*. It has been possible to re-program the frequency imprints of a cell culture and have these were transmitted correctly to cultured daughter cells which demonstrates that lasting effects are possible. The presence of frequencies which fluctuate to a limited extent (a few percent) over time is a sign of a normal healthy biological system. Chemical contamination restricts this activity by imprinting a chemical signature frequency. After a patient has been chemically detoxified, a "memory" or "miasm" of the toxin may remain in the body and this needs to be removed.

3. Entrainment of Environmental Frequencies

There is a surprising degree of interaction between living systems and external frequencies. Although the frequency bandwidth on a meridian is only about $\pm 2\%$ of its mean frequency the latter can be ‘entrained’ or ‘pulled’ by external oscillations such as from an electrical oscillator or an environmental source of radiation such as a computer, TV, mobile phone, or the frequency signature of a chemical. This entrainment may be up to $\pm 30\%$ before the acupuncture meridian frequency jumps back to its normal endogenous value. Table 1 shows this entrainment at the heart acupuncture meridian (He9). The endogenous frequencies were 7.768 Hz and 382 MHz. It should be noted that the 7.8 Hz endogenous frequency of the acupuncture point He9 (also the heart chakra) is exactly 6-times the heart-beat frequency 78/min; it is also one of the frequency bands in the Schumann Radiation from the upper atmosphere.

Table 1

Entrainment by Environmental Frequencies

Exposure Frequency	He9 High Band	He9 Low Band
MHz	MHz	Hz
No Exposure	382	7.768
260	382	7.718
270	270	5.245
370	370	7.652
390	390	7.864
400	400	7.933
450	450	9.830
480	480	7.657
500	382	7.660

The subject was exposed to the high frequency only by sitting in front of the output loop of a microwave oscillator for 3 minutes after which the frequencies on acupuncture point He9 were immediately imprinted into water in a pipette and measured. The pipette tip was placed on the point and a magnet brought close to imprint. The microwave power density at the subject was estimated to be of the order of mW/m^2 . The frequency measurements took about 5 minutes following the exposure by which time the

acupuncture point frequency had relaxed to its unexposed value so another measurement was possible. Table ** shows that at 260 MHz and at 500 MHz there was no entrainment. From 270 MHz to 480 MHz, the frequencies measured on He9 had become entrained to the exposure frequency and the low band frequencies had also shifted in proportion. The frequencies where entrainment has occurred are shown red. Within entrainment, the high-band to low band frequency ratio is: $50.8 \pm 4.7 \times 10^6$ (SD $\pm 9\%$).

This is an example of the “Multiple Frequency Effect” characteristic of a coherent system where the constant parameter becomes the coherence length, this determines the wavelength. This makes frequency proportional to the velocity with which the coherence travels. Any velocity that the system will support has its corresponding frequency, this makes frequency a **fractal** quantity. Table 2 shows this effect for the optical spectrum of mercury imprinted into water; there are additional bands of frequencies in the microwave region and at low frequencies (ELF). It is this which couples the electromagnetic effects of environmental chemicals, microwave radiation to the endogenous frequencies of living systems.

Table 2
Spectrum of Mercury Imprinted into Water -
Showing Multiple Frequencies Fractal Effect

Hg lines	Optical		Microwave		ELF
nm	Hz		Hz		Hz
	$\times 10^{15}$	$\times 10^6$	$\times 10^6$	$\times 10^6$	$\times 1$
185	1.62		935		19.31
254	1.18		680		14.38
365/6	0.820		472		9.843
405	0.740		425		8.925
436	0.688		396		8.358
492/6	0.607		347		7.235
546	0.549		315		6.633
577/9	0.519		298		6.262
615	0.488		280		5.832
623	0.482		276		5.832
Ratio		1.7340		47.70	
Std. Dev.		$\pm 0.34\%$		$\pm 0.75\%$	

4. Frequency Measurements on Patients

The procedures adopted for testing patients for electrical sensitivities are described in the appended “Notes for Patients” and in papers cited in the bibliography. When we started patient testing, we did not know what to expect. It was sufficient for the patient to sit in the same room as a set of electrical oscillators which were tuned slowly over a wide range of frequencies and the clinician noted the frequencies at which symptoms occurred and at which they were neutralized. Subsequently, patients came in who were so sensitive that they could not tolerate an oscillator being switched on anywhere in the building. For these, it was necessary to have the patient hold a vial of water held in the fist and success it on a wooden surface. This imprinted the body fields and frequencies into the water which could then be measured in the absence of the patient. It is just possible to measure such frequencies by instrumentation in the kilohertz region with electrodes or by heats of mixing (V. Elia, M. Niccoli, “Thermodynamics of extremely dilutes aqueous solutions”, *Ann NY Acad Sci* 1999; **879**:241-8). These methods are only useful for validation. The only practical method for clinical purposes is the dowsing technique (Smith, 2004). Thus, allergists like civil and mining engineers may have to be told to go and learn to dowse!

We found that about 10% of patients with chemical, nutritional or particulate sensitivities had acquired electromagnetic sensitivities. The frequencies measured for triggering the reactions or neutralizing them covered a wide range but showed little recognizable pattern until it was realized that 7.8 Hz often appeared. This frequency is used in some therapeutic or protective devices to stimulate the heart meridian. Measurements quickly revealed that each acupuncture meridian (and the Chakras) had a characteristic endogenous frequency (see Tables 2 & 3) and that many of the frequencies measured from these patients were those of the acupuncture meridians. Such measurements show those acupuncture meridians which are under stress and those which need stimulation.

Figure 1 summarizes the frequency imprinting by 12 electrically hypersensitive patients who during the course of their therapy had imprinted a total of 57 tubes of water with a total of 726 frequencies. Of these, 167 would have been capable of synchronization at a Ting acupuncture point, and 655 would have been capable of entrainment. Many patients had more than one frequency capable of entraining St45, hence the >100% values. There were only 49/726 frequencies outside any entrainment range. Ten patients who lived in the EU had imprinted 19/54 tubes with the 50 Hz power supply frequency. Two patients who lived in N. America had imprinted 3/5 tubes with their 60 Hz power supply frequency (nothing at 50 Hz). It appears that adaptation and entrainment to the power supply frequency is quite common among such patients.

The endogenous frequencies on acupuncture points and meridians can be followed right through to the target organ tissue itself. Frequencies were measured in histological microscope slides of the acupuncture target organ tissues where these were available. The paired-values correlation coefficients for classical points vs. target organs were:

Low frequency band:	0.9999
High frequency band:	0.9771

The acupuncture meridians can be stimulated through the eye by looking at a flashing light. A light-emitting-diode was connected to an oscillator and was viewed towards its lens and at a comfortable brightness which did not give any noticeable after-image. The chakra and acupuncture points were checked for reactions when this was viewed at the frequency known to stimulate the particular chakra or meridian. All the reactions measured were of stress with both eyes open. There was only a reaction of awareness when viewing with either eye alone. With both eyes closed there was no response. All the chakra points could be stimulated. The only Ting acupuncture meridians not so affected were the kidney meridian at Ki1 (endogenous frequency 47 kHz) and spleen-pancreas meridian at Pn1 (endogenous frequency 0.05 Hz).

The endogenous frequencies on an acupuncture meridian are very precise. For 31 TW1 frequencies from 22 patients, the mean was 6.0178 kHz (S.D. \pm 0.20%) and for 53 He9 frequencies from 38 patients, the mean was 7.7877 Hz (S.D. \pm 0.92%).

Table 2

Ting Acupuncture Points (after Dr. Voll)

These points are located on the skin at either corner of the nail bed

<u>Points on Hands</u>	<u>Location</u>	<u>Target</u>
	<u>Organs</u>	<u>Acupuncture Points</u>
<u>Thumb</u>		
Outside	Lymphatic tissue, Lungs	Ly1
Inside	Lungs	Lu1
<u>Index Finger</u>		
Outside	Large intestine	LI1
Inside	Nerve degeneration	ND1
<u>3rd. Finger</u>		
Outside	Circulation, Pericardium	Ci9
Inside	Allergy	AD1
<u>4th. Finger</u>		
Inside	Organ degeneration	Or1
Outside	Triple Warmer, Endocrine	TW1
<u>Little Finger</u>		
Inside	Heart	He9
Outside	Small intestine	SI1
<u>Big Toe</u>		
Inside	Spleen, Pancreas	Pn1
Outside	Liver	Liv1
<u>2nd. Toe</u>		
Inside	Joint degeneration	JD1
Outside	Stomach	St45
<u>3rd. Toe</u>		
Inside	Fibroid degeneration	FibD1
Outside	Skin degeneration	Sk1
<u>4th. Toe</u>		
Inside	Fatty degeneration	FatD1
Outside	Gall bladder	GB44
<u>Little toe</u>		
Inside	Kidney	Ki1
Outside	Bladder (urinary)	BL67

Table 3**Acupuncture Points and Nominal Values for their Endogenous Frequencies**

‘Classical’ Acupuncture Meridians	Point Measured	Low Band Frequency	High Band Frequency
		Hz	MHz
Lung	Lu1	0.48	24
Large Intestine	LI1	0.055	2.7
Stomach	St45 / right	0.044	22
Stomach	St45 / left	0.44	2.2
Spleen	Pn1	0.055	2.7
Heart	He9	7.8	380
Small Intestine	SI1	0.025	1.2
Urinary Bladder	BL67	5.5	270
Kidney	Ki1	0.00095	0.047
Pericardium	Pe9	0.25	13
Sanjiao (TW)	TW1	6000	300,000
Gall Bladder	GB44	0.05	2.46
Liver	Liv1	4.8	240
Du Mai (GV)	GV14	4.3	149
Ren Mai (CV)	Ren24	14	730
‘Extra’ Points			
Anmian I & II	Ex 8 & 9	3,000	
Extra ‘Ting’ Points			
Lymphatics	Ly1	0.06	2.95
Nerve Degeneration	ND1	0.00055	0.027
Allergy	AD1	2	98.4
Organ Degeneration	Or1	0.078	3.85
Fatty Degeneration	FatD1	0.74	36
Skin Degeneration	Sk1	0.0035	0.172
Joint Degeneration	JD1	0.3	148
Fibroid Degeneration	FibD 1	800	39,400
Circulation, pericardium	Ci9	0.05	2.46

5. **Relation between Acupuncture Meridians and the ANS**

The relationship between the acupuncture meridians and the autonomic nervous system (ANS) comes from the work of Dr. Reinhardt Voll. In his work, cited in English by Kenyon (J.N. Kenyon, "Modern Techniques of Acupuncture" Vol. 3, Chapter 11 – Disordered Autonomic Steering), Voll identifies a complete system of acupuncture points which indicate the functioning of both branches of the autonomic nervous system. These are listed in Table 4. These points, Voll accessed by his method of electroacupuncture (EAV). He found a drop in the electroacupuncture reading where there was stress on the corresponding part of the ANS.

To be able to relate the results of Voll to the writer's measurements of frequency it was decided to compare the percentage changes observed during electroacupuncture measurements with the resulting percentage frequency changes on the acupuncture meridians.

These measurements (Table 5) were made on Voll's summation point for the entire ANS (nerve degeneration meridian, ND1) on 7-days between February 15 & March 6, 2005 by the writer on himself. The measurement order was: RH frequencies, EAV % change measurement, RH frequencies; LH Frequencies, EAV % change measurement, LH frequencies. There were three frequencies to be measured in each water imprint - the frequency characteristic of acupuncture meridian ND1 ($\sim 4 \times 10^{-4}$ Hz), the frequency characteristic of the sympathetic ANS ($\sim 3 \times 10^{-3}$ Hz) and the frequency characteristic of the parasympathetic ANS ($\sim 3 \times 10^{-1}$ Hz). These frequencies appear at those acupuncture points linked to the ANS in addition to the endogenous meridian frequency.

The frequency changes arose from the electrical stress imposed by the electroacupuncture measurement. The percentage frequency changes approximate to the percentage changes in the electroacupuncture readings. In practice, it is quite difficult to read the electroacupuncture meter needle movement as it takes place while the probe is being applied to the point. Mostly, there was a drop in the acupuncture reading but, where the acupuncture reading showed an increase, the frequency also increased. Measuring the frequencies characteristic of the sympathetic and the parasympathetic systems does appear to indicate which system is under stress on either side of the body.

Table 4

	↓—————	ND1a Nerve Degeneration ANS	—————↓	
St10a	Parasympathetic		Sympathetic	GB20
GB11b	Vagus nerve nucleus in medulla		Sympathetic nerve - cranial	GB19a
St8d	Vagus nerve - cranial		Sympathetic nerve - cervical	GV16
St8c	Vagus nerve - cervical		Cervical ganglion	TW1a
St16	Pharangeal plexus		Sympathetic trunk – thoracic	BL16*
St15	Oesophageal plexus / Vagus thoracic		Sympathetic trunk – abdominal	BL24**
St18	Pulmonary plexus		Coeliac plexus	St44c
St20 L/R	Gastric plexus – anterior/posterior		Sympathetic - Pelvic	BL33
Ki20	Vagus nerve - coeliac		Inferior hypogastric plexus	BL63***
Ki21	Vagus nerve - hepatic			
Ki 19	Vagus nerve - renal			
BL35	Sacral preganglion fibres			
BL34	Pelvic plexus			
BL32	Pelvic splanchnic nerves			

EAV points Indicating the Functioning of the ANS

Notes

***BL16 is the EAV summation point for:**

Ci8e/L	Thoracic aortic plexus
Ci8e/R	Cardiac ganglia
He8e	Cardiac plexus
Lu10d	Coronary plexus
Lu9a	Bronchial plexus

****St44c is the EAV summation point for:**

St19	Phrenic plexus
Ki1b	Supra renal
Ki1d	Renal plexus
St30a	Testicular or ovarian plexus
St22/R	Superior gastric plexus
GB43c	Hepatic plexus
SI1a/R	Superior mesenteric plexus
SI1a/L	Inferior mesenteric plexus
Ci8a	Abdominal aortic plexus
LI1a/L	Iliac plexus
LI1a/R	Superior hypogastric plexus

*****BL63 is the EAV summation point for:**

Ki4	Renal or haemorrhoidal plexus
BL66c	Vesical plexus
BL49d	Prostatic plexus in male / uterovaginal plexus in female
BL50	Cavernous plexus of penis or clitoris.

After Dr. R. Voll from J.N. Kenyon, "Modern Techniques of Acupuncture", Vol. 3, Chapter 11.

Table 5

Frequency Changes on ND1 Following Electroacupuncture Measurements

RH before	RH after	% mean frequency change	% EAV change	LH before	LH after	% mean frequency change	% EAV change
Hz	Hz	ND1		Hz	Hz	ND1	
4.7542 $\times 10^{-4}$	4.5910 $\times 10^{-4}$	3.49 %	5.29%	4.8187 $\times 10^{-4}$	4.5839 $\times 10^{-4}$	2.50%	6.71%
\pm 1.93%	\pm 4.66%			\pm 5.54%	\pm 3.84%		

		Sympathetic				Sympathetic	
3.2485 $\times 10^{-3}$	3.1975 $\times 10^{-3}$	1.48%		3.2353 $\times 10^{-3}$	3.1198 $\times 10^{-3}$	3.63%	
\pm 2.32%	\pm 2.19%			\pm 2.73%	\pm 4.27%		
		Parasympathetic				Parasympathetic.	
3.2133 $\times 10^{-1}$	3.1660 $\times 10^{-1}$	4.73%		3.2169 $\times 10^{-1}$	3.1927 $\times 10^{-1}$	0.76%	
\pm 3.89%	\pm 3.79%			\pm 4.02%	\pm 4.94%		

6. Magnetic Resonance in Acupuncture Meridians

Once it was established that the acupuncture meridians carried characteristic endogenous frequencies, it was clear that a precise magnetic field (determined by the physical constants for the electron or proton) could also stimulate a meridian and thence the ANS through the linkages found by Voll. Those fields above the geomagnetic field are listed in Table 6; proton magnetic resonances are shown in italics. We had previously found that living systems could react to the magnetic resonance conditions even in fields as weak as the geomagnetic field (Jafary-Asl et al., 1983; Aarholt et al., 1990).

Table 6
Magnetic Fields Exciting Resonances on Acupuncture Meridians

‘Classical’ Acupuncture Meridians	Point Measured	Low Band Frequency	Resonance Magnetic Field	High Band Frequency	Resonance Magnetic Field
		Hz	Gauss	MHz	Gauss
Lung	Lu1	0.48		24	8.56
Large Intestine	LI1	0.055		2.7	0.96
Stomach	St45 / right	0.044		22	7.85
Stomach	St45 / left	0.44		2.2	0.79
Spleen	Pn1	0.055		2.7	0.96
Heart	He9	7.8		380	137
Small Intestine	SI1	0.025		1.2	0.43
Urinary Bladder	BL67	5.5		270	96
Kidney	Ki1	0.00095		0.047	<i>11.2</i>
Pericardium	Pe9	0.25		13	4.64
Sanjiao (TW)	TW1	6000	<i>1.42</i>	300,000	
Gall Bladder	GB44	0.05		2.46	0.89
Liver	Liv1	4.8		240	8.7
Du Mai (GV)	GV14	4.3		149	53
Ren Mai (CV)	Ren24	14		730	261
‘Extra’ Points					
Anmian I & II	Ex 8 & 9	3,000	<i>0.74</i>		
Extra ‘Ting’ Points					
Lymphatics	Ly1	0.06		2.95	1.05
Nerve Degen.	ND1	0.00055		0.027	6.43
Allergy	AD1	2		98.4	3.51
Organ Degen.	Or1	0.078		3.85	1.37
Fatty Degen.	FatD1	0.74		36	12.90
Skin Degen.	Sk1	0.0035		0.172	40.9
Joint Degen.	JD1	0.3		148	53
Fibroid Degen.	FibD 1	800	<i>0.19</i>	39,400	140
Circulation	Ci9	0.05		2.46	0.89

Proton magnetic resonance fields are shown in italics.

The other fields are for electron spin resonance.

The small intestine meridian is permanently stimulated by the normal geomagnetic field.

7. Chemical Frequency Signatures and the ANS

Figure 2 shows the frequency signatures for a number of toxic environmental chemicals measured in the blood of 22 EM sensitive patients. The frequencies entrained by the patients correlate exactly with those of the chemical signatures. The blood levels (where available) are the average of values of measured concentrations which varied by up to a factor of ten between different patients.

Figure 3 relates to a patient who was one of about 200 persons chronically exposed to a toxic chemical in their living environment. It was acquired in following manner: In a new building, the waste steam from the heating boiler was designed to be used to humidify the air in the air-conditioning system. In operation, an anti-corrosion and de-scaling chemical product was put into the boiler; this vaporised into the steam and was circulated throughout the building. The chemicals' signatures contained 31 frequencies. A particular patient's body field had 21 frequencies of which 10 corresponded exactly to those of the chemicals as shown in Figure 3. This represents an almost 50% entrainment of a patient's endogenous frequency activity by an identifiable chemical contamination in the building, namely cyclohexylamine and morpholine. Of the 10 frequencies common to the chemicals and the patient, any of the lowest three would entrain the allergy meridian, the next three would entrain the heart meridian which also relates to the cardiac plexus, and all but one would entrain the Du Mai meridian which relates to the status of the cerebro-spinal fluid and to the cervical part of the sympathetic chain.

In general, all biological cells are capable of emitting an electrical signal in response to a chemical stimulus and releasing a chemical in response to an electrical stimulus. Figure 4 maps the pathways for this activity and lists Voll's acupuncture points through which they would link to the ANS. The heavy arrows indicate the emission or absorption of a chemical, the lines indicate electrical pathways.

Tables 7 and 8 show some of the homoeopathic potencies which stimulate the ANS in the sympathetic and parasympathetic branches and the various acupuncture point linkages affected. Those potencies which affected the greatest number of acupuncture-ANS linkage points were selected from among potencies which the writer happened to have available.

Table 7

Homoeopathic Potencies which Stimulate the Sympathetic ANS

	Sympathetic GB20	Sympathetic - cranial GB19a	Sympathetic - cervical GV16	Cervical ganglion TW1	Sympathetic - thoracic BL16
Arsenicum alb. 1M	X	X	X	X	
Lycopodium 6C	X	X			
Chamomilla 30C	X	X	X		
Ac. fluor. 6C	X	X	X	X	
Crotalus 6C/12C			X		X
Electricitas 200C			X	X	
X-ray 200C	X	X			
Carcinosin 200C	X	X			X
CA colon 200C	X	X			X
Petroleum 30C					
Rad. Brom. 1M	X				

	Sympathetic - abdominal BL24	Coeliac plexus St44c	Sympathetic - pelvic BL33	Inf. Hypo-gastric plexus BL63
Arsenicum alb. 1M	X			
Lycopodium 6C	X	X	X	
Chamomilla 30C			X	
Ac. fluor. 6C				
Crotalus 6C/12C		X		
Electricitas 200C	X			
X-ray 200C		X		
Carcinosin 200C				
CA colon 200C		X		X
Petroleum 30C		X	X	X
Rad. Brom. 1M	X			X

Table 8

Homoeopathic Potencies which Stimulate the Parasympathetic ANS

	Para-sympathetic St10a	Preganglion fibres mid-brain GB 10a	Vagus nucleus in medulla GB 11b	Vagus-cranial. /cervical St8 c/d	Pharyngeal plexus St 16	Oesophageal plexus St 15	Pulmonary plexus St 18
Arsenicum alb. 1M				x		x	
Graphites 10M	x		x	x			
Cu. met. 6X		x	x		x		
Carcinosin 200C	x				x		x
Phosphorous 6C		x					
Electricitas 200C	x		x				
Crotalus 6C/12C	x						
Rad. iod. 200C				x			
Rad. brom. 1M		x					

	Vagus – abdominal St 21	Gastric plexus ant. St 20/L	Gastric Plexus post. St 20/R	Vagus- coeliac Ki 20	Vagus- hepatic Ki 21	Vagus- renal Ki 19	Sacral pregang. fibres BL 35	Pelvic plexus BL 34	Pelvic – splanchnic nerves BL 32
Arsenicum alb. 1M		x	x	x	x	x			
Graphites 10M							x	x	x
Cu. met. 6X	x		x						
Carcinosin 200C	x	x	x						
Phosphorous 6C	x				x	x	x		
Electricitas 200C					x	x		x	x
Crotalus 6C/12C	x			x	x	x	x	x	x
Rad. iod. 200C		x	x	x	x	x		x	x
Rad. brom. 1M				x	x	x			x

ANS active Chemical

Table 9 lists as an example the frequencies of a toxic chemical (glyphosate) for which the frequency signature is capable of linking to the whole of the ANS through ND1 (Voll's ANS summation acupuncture point) with further links to the sympathetic and parasympathetic branches through their specific frequencies. In addition there is linkage through the gall-bladder meridian to the preganglion fibres from mid-brain, the nucleus of vagus in medulla and the hepatic plexus; through the circulation meridian to the thoracic aortic plexus, the cardiac ganglia and the abdominal aortic plexus; through the Du Mai (GV) meridian to the cervical part of the sympathetic chain; through the Sanjiao meridian ('triple-warmer') to the cervical ganglion. The $\uparrow 5.011 \times 10^{-2}$ Hz might also be able to entrain the LI meridian which relates through the ANS to the iliac plexus and the superior hypogastric plexus. The fibroid degeneration frequency might be a link to connective tissue.

In general, a chemical frequency signature locks up that particular frequency within the affected part of the living system and prevents normal fluctuations in response to the demands of metabolism or the environment at that frequency.

Table 9
Herbicide (glyphosate)

= stimulatory (hyperactive) \downarrow = depressive or stressful (hypoactive).

Hz	Acu. Meridian	Effect on ANS
$\uparrow 4.536 \times 10^{-4}$	ND	ANS summation point
$\downarrow 3.617 \times 10^{-3}$	Sympathetic branch	Sympathetic branch
$\uparrow 5.011 \times 10^{-2}$	GB,	Preganglion fibres from mid-brain, Nucleus of vagus in medulla Hepatic plexus
$\uparrow 5.011 \times 10^{-2}$	Ci	Thoracic aortic plexus Cardiac ganglia Abdominal aortic plexus
$\downarrow 3.516 \times 10^{-1}$	Parasympathetic branch	Parasympathetic branch
$\uparrow 4.311 \times 10^0$	GV	Cervical sympathetic
$\downarrow 5.000 \times 10^{+1}$	50Hz (tech)	
$\uparrow 8.031 \times 10^{+2}$	FibD	
$\downarrow 6.051 \times 10^{+3}$	TW	Cervical ganglion
$\uparrow 3.05 \times 10^{+5}$		

Note: Plants have a 'meridian' it runs from leaf nodes, to root nodes, to the root tips.

Some Definitions

Waves - Regular or periodic variations or pulsations in space and/or time; their shape is the *waveform* (e.g. sinusoidal, rectangular, triangular, pulse).

Frequency - The number of cycles of regular or periodic variations per second of some parameter. An oscillator is a generator of frequency.

Period - The time between two adjacent corresponding points on a waveform, the reciprocal of the frequency is the period.

Wavelength - The distance in space between two adjacent corresponding points on a waveform.

Amplitude - The maximum, zero-to-peak, value of the oscillating parameter. Amplitude squared is the intensity and is proportional to power. The root-mean-squared (r.m.s.) value is $1/\sqrt{2}$ of the peak value, it delivers the same power as a steady current or voltage having numerically the r.m.s. value.

Phase - The fraction of a complete cycle measured in degrees or radians (1 cycle = 360° or 2π radians).

Velocity of a wave Velocity equals frequency times wavelength (metres/sec = cycles/sec \times metres/cycle).

Coherence - is an expression of the degree of constancy of phase, as for example between two oscillators or waves of nominally the same frequency. The bandwidth divided by the frequency is a measure of the extent to which perfect coherence (zero bandwidth) is achieved in a practical situation. *Coherence Length* is the distance over which the coherence is maintained. *Coherence Time* is the time for which the coherence persists.

Electric Charges and Electromagnetic Waves

Electrostatics describes the properties of electric charges (e.g. electrons or ions) at rest. These charges arise from the structure of matter and the chemical bonds by which matter is condensed from gas to form a solid or liquid. The force on a given charge due to other nearby charges is the measure of the *electric field* in which it is situated. The work done

by this force if the charge moves is its *electric potential*. Magnetic fields have an analogous set of parameters, they occur when electric charge is in steady motion. If electric charge is accelerated or decelerated, the changes in the associated fields travel out into space at the velocity of light, this is *electromagnetic radiation*. If these changes are periodic at some frequency, a wave of oscillations at this frequency travels out into space with the separation between cycles being the *wavelength*.

Energy in an Electromagnetic Wave

The energy per unit volume of the space occupied by electric and magnetic fields is proportional to the square of the field strength. The power density is that power (energy/sec) crossing one square metre; it is called the “Poynting Vector” and is proportional to the product of the electric and magnetic fields. This applies to most technological oscillations, and it is these electric and magnetic fields which give rise to mechanical effects (electric motor) and thermal effects (electric kettle, microwave cooker).

Quantum Effects

Any material object cannot be sub-divided indefinitely; one must eventually come to its constituent molecules and atoms. Likewise, energy ultimately is packaged into so-called quanta. For a single quantum, the product of its position and momentum or, the product of its energy and time, both have a fundamental (Heisenberg) uncertainty associated with them. These products must be at least be equal to Planck’s Constant h divided by 4π . The energy of the quantum is proportional to its frequency (energy = frequency \times Planck’s constant). A magnetic field is also quantized, a single quantum of magnetic flux equals Planck’s constant divided by twice the electron charge ($\sim 2 \times 10^{-15}$ Wb).

A quantum can be in more than one place or its being found in each condition. The basic unit for computing systems using quanta is called the ‘qbit’; it is unlike the usual binary ‘bit’ (0 or 1) in that it only has a probability of having a particular value somewhere from 0 to 1. Memory in living systems is thought to involve the phase of quantum states as in a quantum hologram (P. Marcer & W. Schempp, “The Brain as a Conscious System”, *Int. J. General Systems* Vol 27(1-3) pp231-248, (1998). This is the only memory system which places the mental image where the object is in space and time (a necessity for all ball games).

These effects involve small probabilities which may only become significant if the frequency is very high, the distances very small or, the perturbing random fields from

thermal vibrations are made very weak by extreme cooling. However, in a system of coherent domains such as the Del Guidice-Preparata model for water, perfect coherence is in theory possible if the system can vary the number of molecules in a domain (instead of the frequency) to accommodate the Heisenberg Uncertainty fluctuations. Many of the frequencies and fields discussed here involve the magnetic vector potential component of the magnetic field. The quantum nature of a living system is confirmed if it reacts to the magnetic vector potential field such as that generated by a toroidal coil. This field only has an effect on the phase of the wave function. Such systems may have the 'Josephson Effect' available; this offers frequency/voltage inter-conversion at 500 MHz/ μ V. All these are so-called "non-thermal" effects.

Electrical Hypersensitivity and Water Phenomena

Presentations at:

International Annual Symposia on "Man and His Environment in Health and Disease" held in Dallas, Texas,

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ELECTRICAL SENSITIVITIES?

Notes for Patients on Electrical Sensitivities

Cyril W. Smith, Ph.D.

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What are Electrical Sensitivities?

Many persons suffer from sensitivities to certain foods and environmental chemicals which cause them discomfort, or even in extreme cases prevent them from functioning in any effective manner. Even the minutest amounts of these substances may on occasions “trigger” reactions which are specific to each individual. Warnings regarding ‘peanuts’ are commonly found displayed on food products. When a reaction occurs, some regulatory system within the body ceases to function, or gives alarm signals calling for an unjustified panic reaction. Usually, it is the autonomic nervous system (ANS) which is the first to become compromised. Since this controls involuntary body functions, any part of the body might become affected which is why such effects do not show up in medical statistics.

Germany has introduced the WHO International Classification of Diseases Code T78.4 for “Chemical-Sensitivity Syndrome Multiple” against which this can be reported and statistics collected. There is no electrical equivalent WHO Classification to date but it would seem reasonable for these cases to be recorded as a complication of multiple chemical sensitivities. Sweden regards electrical sensitivity as a *disability* with implications for public places being made safe for the electrically sensitive.

Persons who already have a number of chemical hypersensitivities on-going are at particular risk of acquiring electrical sensitivities whereby the ANS “triggering effect” transfers from a minute amount of the chemical to some patient specific frequency of electromagnetic field in the environment and usually the same symptoms continue to be “triggered”. It is the frequency that matters once some patient specific threshold of intensity or field strength has been exceeded. The range of coherent frequencies encountered extends from circadian rhythms that is from below a milliHertz through audio- and radio- to microwave frequencies to visible light. These effects are ‘non-thermal’, it is the frequency and in particular the spectral power density, the *watts per cycle of bandwidth* which matters, the more precise the frequency - the less power is needed for an effect.

A therapy for alleviating these reactions is called provocation / neutralization therapy. It was developed from earlier work in the U.S.A. by Dr. Joseph Miller of Mobile, Alabama, and further developed at the Breakspear Hospital, Hemel Hempstead, England by its Medical Director Dr. Jean Monro and at the Environmental Health Center, in Dallas, Texas, by Dr. W.J. Rea who has demonstrated the reality of electrical sensitivities in double-blind trials*. This therapy relies on successive serial dilutions of the substance having in sequence

the effects of stimulating and/or quelling the reactions that they produce. This therapy is not a substitute for eventually reducing the total body loading of triggering substances to a level that the individual can cope with. This is done by simultaneously increasing the rate of detoxification and reducing the rate of toxin intake until the body can function normally. However, while it can produce a more immediate alleviation of the symptoms and thereby assist achieving eventual normalization but, it may not be possible to achieve this without some change in the patient's life-style. It is also labor-intensive and therefore expensive.

When patients have acquired a high degree of sensitivity to many factors in foods and/or the chemical environment (multiple-sensitivities) they are very likely to have acquired an abnormal sensitivity to their electrical environment as a part of this 'package' of symptoms. It is rare to have electrical sensitivities without ongoing chemical sensitivities. This electrical sensitivity can become so severe that a person becomes incompatible with technology and unable to function in the modern environment. Electrical sensitivity is not mutually exclusive of other clinical conditions; it can co-exist with and even trigger physical or mental illness. Electrical sensitivities make diagnosis and therapy more difficult. Medications may produce abnormal responses, side-effects or even chronic sensitization to the electrical environment.

The Electrical Environment

Such patients may experience problems from their natural environment; weather changes, impending weather fronts or thunderstorms may become troublesome; there may be sensitivity to sunlight. Fluorescent lighting may make shopping difficult, particularly if inhalants such as chemicals on fabrics provide the initial sensitization. The patient may experience problems when near electrical equipment, power lines, transmitters, TV's, tape-recorders, computers, mobile phones or one of the many other electronics devices in the modern environment.

The female characteristic is towards chronic sensitivities appearing at an early stage and getting the labeled "neurotic"; the male characteristic is for no reaction until the onset of a sudden and disabling crash in the ability to function.

Persons may even become aware of actually having electrical things malfunction when they handle them or, even when in their vicinity. The hazard of chronic over-exposure to an electrical frequencies is, to use an analogy from homoeopathy, one of '**adaptation**' to '**proving symptoms**' corresponding to a particular pattern of frequencies until they become indistinguishable from the '**disease condition**'.

Typical Symptoms of Electrical Sensitivities include:

Drowsiness, malaise and headache, mood swings, tearfulness and eye pain, poor concentration, vertigo and tinnitus, numbness and tingling, nausea and flatulence, convulsions, noise sensitivity, alteration in appetite, visual disturbances, restlessness, blushing.

Clinical Observations include:

Changes in respiration, heart rate changes (a good indicator of the status of the ANS), eye pupil dilation, perspiration, muscular weakness, loss of visual acuity, speech difficulties, loss of consciousness, convulsions.

Testing for Electrical Sensitivities

Just as abnormal food and chemical sensitivities can be tested for, so can electrical ones. Initially, the procedure was simply to sit the patient in a controlled environment. In practice, this is a chemically and particulate clean room with negligible electrical fields coming in from outside and lit by daylight. An electrical oscillator located at about the distance for TV viewing away from the patient, gives the patient a controlled electromagnetic field comparable with that experienced near a TV or computer.

The person carrying out the test slowly tunes the oscillator(s) through all the environmental frequencies likely to be giving problems. This is usually from below 1 kilohertz (Circadian rhythm frequencies) through 1 Hz (Hz = Hertz = cycles per second) that is around the heart beat and brain wave frequencies, perhaps in rare cases to more than 100 GHz (one-hundred-billion Hertz) far beyond the frequencies of microwave cookers and mobile phones. Some patients are light sensitive.

The patient reports on any symptoms as they may be felt; the tester may also attempt to detect symptoms before they become too uncomfortable for the patient. These symptoms will usually be the same as the symptoms triggered during foods and chemicals testing. This information is available to the tester who needs to know if a heart condition or loss of consciousness is likely to occur. Those frequencies at which the symptoms are “triggered” and “neutralized” are recorded. This method has been used in double-blinded trials to demonstrate the existence of electromagnetic sensitivities with 100% success*.

There are usually one or more of the frequencies at which all the symptoms clear up together. This amelioration will not be maintained if there is a heavy body load of toxic chemicals, environmental or nutritional stresses. However, patients do get great relief in realizing that the symptoms that they have suffered from for years can be turned on/off at will from an electrical oscillator on the other side of the room, not connected to them in any way and that it is not “all in the mind”.

Some patients are so extremely sensitive as to be unable to tolerate frequencies at even TV/computer strength electromagnetic fields. To cope with these cases, a tube of saline or water from any source which is known to be tolerated by the patient, can be given to the patient to hold and preferably ‘succuss’ by banging on a wooden surface. This is then tested away from the patient to find the neutralizing or therapeutic frequencies for this patient at this time. These frequencies can be imprinted into a tube of water to make the equivalent of a homeopathic potency and sent through the mail if wrapped in aluminum foil. With many years experience this is now the writer’s preferred method of testing most patients for frequencies to which they may be sensitive.

These tests often show that certain acupuncture meridians or chakras are under stress. Also, stress may be seen coming from some common environmental frequency such as the 50Hz power supply (60 Hz in North America).

Sensitivities to Foods and Chemicals:

Severely electrically sensitive patients are likely to have responses to chemicals and other factors in the workplace. About 1-in-6 of a population has some impaired function due to some allergic reaction to the environment or to food. Repeated exposure to a given frequency, while a person is reacting severely to some different allergic trigger can frequency sensitize that specific sensitivity pattern so that the same reaction is triggered on encountering that frequency subsequently. In general, the pattern of response is the same whether the trigger is chemical, biological, particulate, nutritional or electrical – *it is characteristic of the patient.*

Exposure to pesticides or herbicides seems to enhance or even create electrical sensitivities. Ionizing radiation exposure appears to represent an additional stress factor. A few persons become hypersensitive to light, some to daylight, but particularly to the coherent light of the mercury vapor spectrum which is superimposed on light from fluorescent tubes.

Dental fillings may cause problems due to electrolytic currents between different types of amalgam in the teeth, between fillings and surrounding tissue or from environmental frequencies rectified at metallic contacts.

Treatment:

There is an effective treatment for many allergic responses to foods, chemicals and inhaled matter. This includes neutralizing the effects of problem foods and chemicals, minimizing exposure to electromagnetic frequencies and noxious chemicals, restoring nutritional status especially of cell membranes and the removal of heavy metals. The general concept introduced by Dr. W.J. Rea is to seek to *reduce the total body load of stressors*. Which stress factors one seeks to reduce may be a matter of choice although some stresses are involuntary. Where chemical stress already exists exposure to any electrical stress may not be an option.

As the foods and chemicals sensitivities are brought under control and the body de-toxifies itself, the electrical sensitivities usually disappear as well. However, it is worth noting that if a person is working or sleeping in a zone of "geopathic stress", which may be electrical in origin, then their problems may persist and resist therapies. Symptoms usually disappear in the reverse order to their appearance.

Scientific Background

The bio-effects which can be described by "*classical physics*" are the effects of heat or mechanical force; it cannot even account for chemistry. Yet, this is the model used in EM risk-assessment. As soon as it becomes necessary to consider the effects of energy coming in packets or 'quanta' then one has arrived at

“*quantum physics*”. I presented evidence for living systems behaving as large scale quantum systems in a lecture to The Frontier Sciences Department of Temple University in Philadelphia in 1997. (Smith C.W. “Is a living system a macroscopic quantum system?”. *Frontier Perspectives*, 7(1), 9-15 (1998).

The important aspects of the science involved are:

1. **Frequency:** is the quantity of prime importance not intensity.
2. **Coherence:** is the precision of the frequency and its phase.
3. **Coherence Length:** is the distance over which coherence is maintained - the coherence domain.
4. **Multiple Frequencies Effect:** within a coherence domain the constant parameter is the coherence length so, the velocity with which the coherence travels becomes proportional to its frequency. There can be many velocities and corresponding frequencies and all can interact. It is this which brings the frequencies and effects of chemical reactions down into the technological range of frequencies. If there was not a duality between chemical structure and frequency, spectroscopic analysis would be impossible.
5. **Magnetic Fields:** there is a component of a magnetic field called the *magnetic vector potential*. It is in the direction of the current giving rise to the magnetic field and cannot be shielded by usual methods. It can give biological effects at the quantum level. By altering the phase of the wave function (as can an electrical or chemical potential).
6. **Memory:** recent research is showing that memory works like a hologram storing information as the phase of a frequency rather than as in a digital computer memory. The whole of a mental image can be displayed or processed in one go, it is not built up like a television picture from a moving spot. The ‘quantum’ aspect in holography means that instead of memory locations being empty or full (‘0’ or ‘1’) there is only a *probability* for each state being ‘0’ and ‘1’. Chemical memory (like DNA) is also thought to work like this. There is a syntax structure as in a language or a computer re-write program. Non-local effects as in quantum theory may be expected.

EM Sensitive Patient Support Groups

There are a few ‘EM Sensitive Patients’ Support Groups’ around, these are often run on a voluntary basis by persons who themselves are highly electromagnetically sensitive. It is usually necessary to contact them by mail as they may be unable to use a computer, email or telephone.

‘CIRCUIT’ - Electrical Sensitivity Support Group, P.O. Box 1UZ, Newcastle-upon-Tyne, NE99 1UZ, England. www.ukindex.info/circuit

IERVN, P.O. Box 231, Sorting Office, Cork, Ireland.

ADECEM, 55 bd. de Strasbourg, F-75010 Paris, France.

Selbsthilfverein für Elektrosensible e.V.
Gesundheitshaus der Stadt München, Dachauer Str. 90, D-80335 München,
Germany.

Arbeitskreis für Elektrosensible e.V. Aleestrasse 135, D-44798 Bochum,
Germany.

Selbsthilfegruppe Elektrosmog Bayreuth, Dipl. Ing. Franz Mayerhofer, Ringhau 1,
D-95515 Plankenfels, Germany.

El-og Billedskaermsskadede i Danmark, PO Box 88, 3000 Helsingør, Danmark.

FEB, Box 151 26, 104 65 Stockholm, Sweden.
Information sheets: "Electrical Hypersensitivity", "Technical Guide for the
Electrically Sensitive"

Association for the Electrically Hypersensitive, c/o Per Hysby, Stubbanv. 2, 7037
Trondheim, Norway.

ERGOTEC, PO Box 9571, Arlington VA 22209, USA.

National EMR Alliance, 410 West 53rd. St. Suite #402, New York NY 10019,
USA.

Microwave Awareness Group of Yelm, PO Box 1384, Yelm WA 98597, USA.

Cellular Phone Taskforce, PO Box 1337, Mendocino CA 95460, USA.

Electromagnetic Research Foundation, c/o Dr. Duane Dahlberg, 1317 Sixth Ave.
N., Moorhead MN 56560, USA.

Disability Council of the White Mountains, c/o Susan Molloy, PO Box 483,
Snowflake AZ 85937, USA.

EMR Network , Janet Newton – President, PO Box 221, Marshfield VT 05658,
USA.

Electromagnetic Radiation Task Force Canada, Milt Bowling – Director, 3570
Corsica Way, Vancouver BC, V5S 4J3, Canada.

EMFacts Consultancy, PO Box 96, North Hobart, 7002 Tasmania, Australia
E:mail: dmaisch@emfacts.com
Web: <http://www.emfacts.com>

Klaus Rudolph - Citizens' Initiative Omega Newsletter Star.Mail@t-online.de

Useful Websites About EMF

- U.K. - www.em-hazard-therapy.com Newsletter
www.powerwatch.org.uk EMF measurements and
equipment
- U.S.A. - www.aehf.com American Environmental Health
Foundation
- Sweden - www.microwavenews.com
www.feb.se
www.isy.liu.se/~tegen/febost.html

Further Reading

For more about the whole subject of health and hazard in our modern electrical environment, try the non-technical parts of the book: "Electromagnetic Man" by Cyril W. Smith and Simon Best; it was published in 1989 & 1990 by J.M. Dent, Ltd., London. It is still out-of-print but there are copies in the U.K. public libraries system.

The paper by CW Smith on "Nursing the Electrically Sensitive Patient" in: *Complementary therapies in nursing & midwifery* **3**: 111-116 (1997) and the Guest Editorial "Energy Medicine United" **9**: 169-175 (2003) and might also be useful.

There is also, "The Electrical Sensitivity Handbook" Lucinda Grant, Weldon Publishing, and Prescott AZ (1995) ISBN 0-9635407-2-6.

*Double-Blind Trial

For evidence that electromagnetic field sensitivity actually does exist and can be elicited under environmentally controlled double-blind conditions with 100% reactions to an active frequency and 0% to the placebos, see: Rea WJ. Pan Y. Fenyves EJ. Sujisawa I. Suyama H. Samadi N. and Ross GH. "Electromagnetic Field Sensitivity", *Journal of Bioelectricity* **10(1&2)**: 241-256 (1991).

Objectives & Notes

Martha Stark, M.D.

Date of talk: Friday, June 10, 2005, 10:30am

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Training:

Current Job Description: Psychiatrist/Psychoanalyst; Faculty, Boston Psychoanalytic Institute; Faculty, Massachusetts Institute for Psychoanalysis; Faculty, Center for Psychoanalytic Studies, Massachusetts General Hospital

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Medical School/ University Attended Harvard Medical School

Internship:

Residency: Adult Psychiatry Residency, The Cambridge Hospital, Cambridge, Massachusetts, Child Fellowship, Massachusetts Mental Health Center, Boston, Massachusetts

Board Certifications: American Association of Psychiatric Medicine

Other Information: Working with Resistance (1994). Northvale, NJ: Jason Aronson.
A Primer on Working with Resistance (1994). Northvale, NJ: Jason Aronson.
Modes of Therapeutic Action (1999); Northvale, NJ: Jason Aronson.

SPEECH TITLE: “The Synergy of Mind and Heart”

At the end of this Presentation, the participant should be able to:

1. Appreciate the ways in which heart rate variability offers a dynamic window into autonomic function.
2. Understand the importance of balancing an overstressed autonomic nervous system.
3. Recognize the healing techniques used to balance the autonomic nervous system.

The above information was provided by the Speaker.

**THE HEART OF THE MATTER
Martha Stark, M.D. - Friday, June 10, 2005**

Abstract:

In order to assess the degree of autonomic nervous system balance, we can use heart rate variability, a non-invasive technology that offers us a dynamic window into autonomic function through measurement of variability (or fixity) in the heart's beat-to-beat (R-R) intervals. With results that are highly reproducible, HRV is a relatively simple test that is an important barometer of the individual's capacity to adapt to stress and other environmental demands and, therefore, a significant indicator of health and predictor of longevity.

In order to determine the variable impact of chest breathing and abdominal breathing on heart rate variability, I conducted an experiment on 36 subjects, using Alexander Riftine's Orthostatic Provocation Test to measure the ability of the ANS to adjust to the "stress" of being challenged to transition from a supine to an upright position. The first measurements were taken when my subjects were breathing normally. For the second set of readings, I "forced" abdominal breathing by instructing them, first, to tighten their Kegel muscles (at the end of each exhalation) and, then, to release those muscles (simultaneously unlocking their diaphragm, triggering a deep, abdominal breath on the next inhalation.

The results of my experiment were hardly definitive, but they did appear to lend support to the idea that the autonomic nervous system, once thought to operate entirely beneath the threshold of awareness, can actually be trained to "learn" from experience, meaning that it is no longer, strictly speaking, autonomic (or self-governing).

**23rd Annual International Symposium
on Man and His Environment in Health and Disease**

THE HEART OF THE MATTER

Martha Stark, M.D.

Friday, June 10, 2005

the **autonomic nervous system** - named "**autonomic**" because it is almost entirely **self-regulating and independent of conscious control**

a **more-or-less closed system** depending primarily upon **innate feedback loops to perform its function of maintaining the body's homeostatic balance** the sympathetic and parasympathetic branches of the ANS operate continuously and, for the most part, **silently (beneath the threshold of awareness) to regulate the ebb and flow of the body's metabolic energy and vital processes**

in and out, expansion and contraction, speed up and slow down, catabolic and anabolic, break down to produce energy and build up to replenish reserves, expend whenever necessary and conserve wherever possible, mobilize and dampen, focus and relax, stimulate and recover

the stress response, the relaxation response

the yang and the yin of energy management

the **stress response** - an **adaptive process** that **begins in the brain** and, by way of the **autonomic nervous system** (which reacts almost immediately) and the **hormonal system** (whose reactions occur more slowly and persist for longer periods of time), releases a **cascade of powerful hormones that prepare the body to deal with the stressor**

a healthy stress response also **includes the ability to recover by activating the other part of the ANS** (called the relaxation response) once the stressor has subsided

of note is that many of the components of the stress response have a **temporary adaptive effect** but a **debilitating effect over time**

in fact, **chronic activation of autonomic tone** leads to **loss of the ability to adapt**

it leads to **rigidity and brittleness, and, ultimately, to exhaustion and breakdown**

the **ANS becomes activated whenever the body encounters something that disrupts its state of internal balance and harmony**, when the body is challenged with stressors

whether internal or external, positive or negative, real or imagined (the body cannot tell the difference)

all are experienced as **perturbations to which the body must adapt in order to restore its balance**

in essence, the **ANS orchestrates the stabilization of our internal environment**
the **internal self-correcting mechanisms** involve **compensatory microadjustments that enable the body to maintain its stability**

depression - may be **secondary to low energy** caused, say, by an **underactive thyroid or exhausted adrenals**

in other words, some depressions (particularly the so-called retarded or depleted depressions) may be the **body's adaptive response to fatigue**, a **slowing down and a withdrawal in order to avoid further energy expenditure and depletion**

correction of the underlying energy problem (through energy work and thyroid or adrenal supplementation) **may then relieve the depression**

the autonomic nervous system is all about homeostasis

rarely, however, is the **distinction** made **between homeostasis as an ongoing process and homeostasis as a state of internal balance achieved as the result of such a process**

the concept of homeostasis, therefore, can signify **both the process of balancing and the achievement of such a balance**

one of the things that makes **chemical sensitivity (and environmental illness)** so frustratingly elusive and difficult for mainstream doctors to understand is the fact that, for the most part, they are **invisible**, involving, as they do, **internal processes that are beyond conscious control, are difficult to comprehend, cannot be seen, and are almost impossible to explain**

chemically sensitive (EI) patients often exhibit a **bewildering and challenging array of symptoms** -- most of which are **largely autonomic in nature, that is, most of which are the result of an autonomic nervous system gone awry, no longer responding in an adaptive way to the vicissitudes of daily life**

patients with EI (and dysautonomia) may experience **rapid and unpredictable fluctuations in their energy levels**, such that they feel exhausted at times when they would want to feel alert and become agitated or restless at times when they would want to relax or sleep

the **"neurasthenic" patients of Freud's day** -- patients suffering from weakness or exhaustion of their nervous systems and often dismissed as attention-seeking hysterics or malingerers -- might actually have been suffering from **EI and a dysregulated ANS abnormal neurovascular activity with cerebral vasospasm**

in any event, **necessary for optimal health is that the autonomic nervous system be able continuously to adapt to whatever demands are made upon it** -- such that a **state of homeostatic balance within the body can be maintained or, if temporarily lost, recovered**

optimal health involves a **healthily functioning autonomic nervous system ever responsive to the constantly changing environmental demands it encounters**

the two branches of the ANS exist in a state of **dynamic equilibrium, opposing but more or less equal "forces" enabling it to operate within an optimal (but tightly regulated) range**

these **microadjustments** are an important part of the **body's generalized adaptation to stress**

the concept of **homeostatic adaptation** speaks, therefore, to **these ongoing compensatory microadjustments initiated by the body's internal regulatory systems (especially the autonomic nervous system) in an effort to maintain the body's stability in the face of stressors threatening to disrupt its homeostatic equilibrium**

it is often assumed that chronically elevated sympathetic tone is unhealthy (which indeed it is); but it has also been generally assumed that increased parasympathetic tone is healthy -- which it is not

in fact, **both "resting" tones should be in mid-range in order for the ANS to respond adaptively and, therefore, optimally**

animals are quite **good at balancing the two branches of their autonomic nervous systems**

healthy cats and dogs will fight hard and run fast but then be able immediately to relax and settle down for an afternoon nap

so, too, **most babies can transition rapidly from a state of intense sympathetic arousal to a state of deep parasympathetic relaxation and sleep**

interestingly, **neuropsychologists** are discovering how **nuances in an infant's autonomic activity are profoundly -- even if subtly -- influenced by its ongoing relationship with its mother**

because of its dependence on its caregivers for environmental and emotional regulation, **if the parenting person can both stimulate and soothe the child, then these regulatory functions will be internalized as capacities in the evolving nervous system of the child**

but if a baby is surrounded by an **intensely conflictual or chaotic environment that chronically overstimulates**, the baby may well respond with **sympathetic overactivation**

or if a toddler's excitement is greeted with **harsh disapproval by a mother made anxious**, the toddler may well respond to this painful withdrawal of loving contact with **parasympathetic overactivation and a drop into shame (that is, a state of collapse, resignation, and internal surrender)**

these **deficits and failures in the child's primary relationships** will be **internally structuralized as "autonomic patterns"** that will become, over time, the **person's default style of coping with emotional stimuli and other stressors**

as another example - if a child never learned from her parent to modulate and temper her more aggressive sympathetic urges, then as an adult she may be unable to engage her parasympathetics to calm herself, resorting instead to impulsive action without much forethought

in fact, more generally, **impulsivity** may well be, in part, a **manifestation of overactivated sympathetics and underemployed parasympathetics**

or, if the two branches of the ANS fail to regulate each other properly, there may be **extreme instability** characterized by wild fluctuations in mood (emotional lability) and transient on-again / off-again body symptoms

but whatever the combination of inborn or acquired, this **stressful modern world** in which we find ourselves living is such that most of us, as adults, have **overactivated sympathetic nervous systems (whereby we feel perpetually wired) and weakened parasympathetics (whereby we feel perpetually tired)**

both situations a result of the **cumulative impact of stressors** (both internal and external, psychological and physical, real and imagined, "too much bad" and "not enough good") **over time**

furthermore, **as we get older, we tend to lose our ability to transition rapidly from one state to the other**; we become **less adept at adapting to stressors**; we have **fewer reserves**; we become **more rigid, more fixed**

because a **chronically overloaded autonomic nervous system leads to loss of plasticity and adaptability**, because it leads to **brittleness, exhaustion, and breakdown of the body systems**, we in the 21st century are now **plagued by a broad spectrum of stress-related diseases with their accompanying autonomic dysfunction** (from chemical sensitivities and food intolerances and environmental allergies to cardiovascular disease and cancer)

when the ANS becomes overstressed, it loses its ability to adapt to stress -- and we become much more vulnerable to chronic, debilitating illness

whereas too much stress is detrimental to health, too little stress can also be unhealthy

it has been suggested that **"...the best cure for a sluggish mind is to disturb its rhythm," which is why challenging an aging brain with word and number problems can help to keep it young**

so, too, **the best cure for a sluggish autonomic nervous system may be to disturb its rhythm -- in order to reinforce its plasticity, its adaptability, its flexibility**

it could be said that **all living systems** operate best at the **interface between sluggishness and disturbance, complacency and arousal, the relaxation response and the stress response, order and chaos -- a delicate balance** the negotiation of which enriches the texture of our lives, enhances the feel of each moment, and promotes optimal health and productivity

in other words, an **optimal level of variability within an organism's key regulatory systems is critical to the inherent flexibility and adaptability that epitomize healthy functioning -- whether of the mind or of the body**

in order to assess the degree of this balance, we can use **heart rate variability, a non-invasive technology that offers us a dynamic window into autonomic function through measurement of variability (or fixity) in the heart's beat-to-beat (R-R) intervals**

with results that are **highly reproducible**, HRV is a relatively simple test that is an **important barometer of the individual's capacity to adapt to stress and other environmental demands** and, therefore, a **significant indicator of health and predictor of longevity**

in essence, heart rate variability refers to the **regulation of the sino-atrial node, the natural pacemaker of the heart, by the synergistic activity of the sympathetic and parasympathetic branches of the ANS**, these two branches in a **constant tug-of-war on the pacemaker**

in **1965**, two obstetricians discovered that **fetal mortality** was **highly correlated with the regularity of the fetus's heart rate** -- the more metronome-like the heartbeat, the less likely the fetus would be to make it; the more variable the heartbeat, the healthier the heart, the healthier the autonomic nervous system, and the more likely the fetus would be to survive

in other words, the outcome was better **the more responsive, the more adaptable, the more variable the heart's beat-to-beat intervals**

respiratory sinus arrhythmia is the **natural variability that occurs as a result of breathing**

every time you **inhale**, your **heart rate increases** because your **sympathetics are stimulated** (temporarily overriding the dampening influence of the vagus on the sino-atrial node); when, inevitably, you then **exhale**, your **heart rate decreases** because the **vagus becomes more activated**

these **cyclic oscillations in your heart rate variability** are normal and healthy

reduced HRV has been linked to almost all the **psychological problems** with which our patients present

a number of studies have documented that **patients with anxiety and panic disorders exhibit a lowered HRV** because of an overactivated sympathetic nervous system that keeps sending out unnecessary warnings of danger or "false alarms"

so, too, **patients with so-called avoidant, dependent, or compulsive personalities** appear to have a low threshold for fear and, as a result, a **heightened arousal of their sympathetic nervous systems**

similarly, **patients with post-traumatic stress disorder**, who are known to have **chronically dysregulated autonomic nervous systems** and to suffer from hair-trigger startle responses, blackouts, flashbacks, hyperirritability, easy distractibility, and disrupted sleep architecture, show **consistently reduced HRV**, even when not exposed to trauma-related incitants

interestingly, **some data suggest a correlation between decreased HRV and depression, but other data suggest no such correlation**

consistent with my hypothesis (which I had offered last year at this conference) that there may be **two kinds of depression (agitated depressions and retarded depressions)?**

representing **different stages in the body's response to stress** and, therefore, **variable degrees of autonomic dysfunction as reflected in variability of impact on the heart rate**

also of note was the finding that **psychotropic medications with anticholinergic side-effects** (tricyclics like nortriptyline -- Pamelor or Aventyl), while still associated with a reduction in depressive symptomatology, actually **reduced heart rate variability**, whereas **selective serotonin reuptake inhibitors** (like paroxetine -- Paxil), equally efficacious antidepressants, were found to have **no effect on HRV**

with respect to different approaches to the treatment of dysautonomia

the use of **breathwork to balance the ANS**
meditating on the breath, so-called **breath awareness**, produces a deeply relaxed state, **decreases the arousal of the sympathetic nervous system, increases the activation of the parasympathetics, and produces alpha brain waves**

breath meditation **trains your conscious mind to focus better and your unconscious mind to relax better**; it **improves your overall relaxation potential and strengthens your powers of concentration**

according to **Herbert Benson**, who coined the term "**relaxation response**" in the **1970s**, if breathwork is to have an impact on your heart rate, then you have to **cultivate a passive mental attitude of surrender (or "letting go")** and you must at least **attempt to achieve focus of your mind**, thereby **freeing it from its distractions**

abdominal (diaphragmatic) breathing is the **natural breathing of newborn babies and sleeping adults**

but the **many stresses of daily living** often put people into a **chest breathing mode** **stress and anxiety lead to a tightening of the diaphragm, and tightening the diaphragm creates further internal tension**

by the same token, relaxation leads to a release of the diaphragm, and unlocking the diaphragm invites relaxation

when the **diaphragm is locked**, then the **upper lungs are selectively filled**; but, because they have more "dead space" (inasmuch as they house the airways through which the air passes en route to the air sacs), the **mechanics of oxygen acquisition will not be all that efficient**

in order to make up for the decreased volume of oxygen acquired with each breath, **chest breathers automatically (autonomically) compensate by increasing their respiratory rate to perhaps 12 to 18 breaths per minute -- which is associated with activation of the sympathetic nervous system**

when, however, the **diaphragm is unlocked** (as happens during diaphragmatic or abdominal breathing), then the **lower lungs can be utilized**, thereby **allowing for an increase in the amount of oxygen inhaled with each breath, much more efficient oxygen consumption, a decrease in the respiratory rate** (to perhaps 6 to 8 breaths per minute), and a **more balanced autonomic nervous system**

in order to determine for myself the **variable impact of chest breathing and abdominal breathing** (and inspired by Alan Vinitzky and Natalie Golos's book entitled Energy -- The Essence of Environmental Medicine), I decided to conduct **my own heart rate variability experiment on 36 hapless "volunteers"** (more or less randomly selected subjects from amongst my friends, colleagues, fellow dancers, and patients)

using **Alexander Riftine's Orthostatic Provocation Test** to measure the **ability of the ANS to adjust to the "stress" of being challenged to transition from a supine to an upright position**, I chose to **focus on the degree of so-called Physical Fitness**, which can **range from an awe-inspiring 1.1 to a terribly compromised 13.7** (reflective of 13 levels of physiological functioning and 7 levels of adaptational reserve)

the **first measurements** were taken when my **subjects were breathing normally**

for the **second set of readings**, I wanted my subjects to try their hand at **abdominal breathing**, although it was difficult for me to teach them to unlock their diaphragms

but then I discovered a way to **"force" them to unlock their diaphragms in order to initiate abdominal breathing**

and so I instructed my subjects to relax, to inhale slowly and deeply through their noses, to exhale slowly through their pursed lips, at the end of which time they were to **tighten their Kegel muscles**, and then, **upon release of those pelvic floor muscles**, once again to **inhale deeply through their noses** -- becoming aware of the expansion of their bellies to accommodate the rush of air into their bodies

I had figured out that **each time the Kegel muscles are released, the diaphragm is also released, thereby triggering a deep, abdominal breath on the next inhalation**

of my 36 subjects, all but 10 improved their overall Physical Fitness levels by a factor of 2 or more, whether with respect to the level of physiological functioning or the level of adaptational reserve

of those 10, 3 became more sympathetically activated, reporting that they had been so intent upon "doing it right" that they had been unable to relax at all; 4 reported that they considered themselves to be, already, abdominal breathers (thus no difference in the before and after); and 3 acknowledged that, when they had been unable to master the Kegels, they had reverted to their normal chest breathing

but with respect to the **remaining 26 subjects**, there were **some rather dramatic improvements in HRV**

GE who went from 11.5 to 8.5

GB who went from 11.4 to 8.4

ES who went from 8.2 to 5.1

JD who went from 5.4 to an impressive 2.2

again, remember, ordinarily, as Rifting can attest to, the **heart rate variability results are very reproducible, remaining very constant over time**

the **results** of my little experiment are **hardly definitive**, but they do appear to **lend support to the idea that the autonomic nervous system, once thought to operate entirely beneath the threshold of awareness, can actually be trained to "learn" from experience, meaning that it is no longer, strictly speaking, autonomic (or self-governing)**

this, of course, is the **basis for biofeedback**, the premise for which is that the **ANS can come under voluntary control through operant conditioning**

a concept developed by **B.F. Skinner** to describe **modification of the body's physiological systems and behavioral responses through positive and negative reinforcement**

and doesn't my little experiment suggest that **if a person has enough discipline to train herself to make a habit of doing deep breathing** (which, of course, is what "conscious breath" and yogic breathing or pranayama are all about), **won't she find herself, eventually, breathing this way much more routinely and having, concurrently, a consistently improved heart rate variability as a result?**

and **if she is able to maintain this consistently improved heart rate variability by way of her abdominal (or diaphragmatic) breathing, doesn't this signify an improved capacity to adapt to stress, a more balanced autonomic nervous system, and prolonged longevity?**

in closing: Antoine de Saint-Exupery's beautiful story of The Little Prince: "And now here's my secret, a very simple secret: It is only with the heart that one can see rightly; what is essential is invisible to the eye."

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Friday, June 10, 2005

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Objectives & Notes

Colin Little, M.D.

Date of talk: Friday, June 10, 2005,
11:00am

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Training:

Current Job Description:	Physician and Allergist, Research participant in CSIRO project on cow's milk intolerance in adults.
Medical School/ University Attended	Melbourne University
Internship:	Western General Hospital
Residency:	Western General Hospital
Board Certifications:	MB, BS, MRCP (U.K.), FRACP, FACA
Other Information:	Current trends in Immunology Vol3 (2000) pgs131-144; Proceedings Soc Exp Biol Med Vol 224 (2000) pgs 264-272; Exp Biol Med (Mini review) Vol227 (2002) pages 438-444; Infect Immun Vol 68 (2000) pgs 3840-47

SPEECH TITLE: “Immune Mechanisms Associated with Cows’ Milk Intolerance”

At the end of this Presentation, the participant should be able to:

1. Appreciate the current research position of milk intolerance.
2. Understand the immunological mechanisms involved.
3. Be aware of the progress and potential of assays of cellular immunity.

The above information was provided by the Speaker.

IMMUNE MECHANISMS ASSOCIATED WITH COWS’ MILK INTOLERANCE

Immune based reactions to cows’ milk may be either immediate or delayed. Immediate reactions are associated with symptoms such as urticaria and wheeze and skin prick tests are positive. This allergy normally disappears by the age of three.

Delayed reactions to cows’ milk are less predictable and more controversial. Skin prick tests to cows’ milk are usually negative and studies suggest the problem persists, at least well into childhood. Associated symptoms include reflux, colic, diarrhea (or constipation) and eczema.

Our research group is studying adults suspected to be intolerant to cows’ milk with delayed reactions. In some cases symptoms have been present since childhood. The patients have had predominantly gut symptoms such as abdominal pain and diarrhea. A double blind protocol has been used for evaluation.

As a starting point for immune evaluation we have drawn on the work of Heyman et al. They studied infants and young children with diarrhea associated with the ingestion of cows’ milk. These young children were skin prick negative to cows’ milk. They demonstrated increased proliferation of white blood cells on culture with cows’ milk proteins and increased production of the cytokine TNF α . The production of TNF α was dose dependent and considerably less when white cells were cultured with digested milk proteins. Their work has been confirmed by an independent study. Other workers have demonstrated increased levels of TNF α in faecal fluid following cows’ milk challenge in milk intolerant children.

This production of TNF α is most probably from TH1 cells. However the earlier studies did not show increased Interferon- γ production. Also the proteins used in these studies were almost certainly contaminated by bacterial lipopolysaccharide (LPS).

We have refined the culture methods and used milk protein free of LPS, and specifically stimulated the differentiation and proliferation of cows' milk-specific T cells. To further modulate the culture system, in some cases anti TGF β antibody (blocks regulatory T cells), TGF β and IL-10 have been added.

Our findings to date indicate that in our patient group there is increased Interferon- γ production (but not IL-5) by peripheral white blood cells on culture with milk protein in comparison with controls. The addition of anti TGF β considerably enhances Interferon- γ production in the patient group but not in the controls and the addition of TGF β and IL-10 substantially reduces interferon γ production.

Our conclusions to date are that controls have regulatory T cells but not effector T cells specific for cows' milk proteins. However the patients have both types of T cells with a predominance of TH1 effector T cells. Methods to efficiently identify the presence of these effector T cells may provide the basis for useful diagnostic test.

It is proposed that the patient group show an aberrant immune response to cows' milk proteins. Effector T cells specific to cows milk migrate to the gut (and sometimes other tissues) where cytokines, including Interferon- γ and TNF α , are released on contact with antigen. A further component of the response may be the possible presence of light chains specific to cows' milk proteins, which are derived from B cells and which recent reports indicate accompany the TH1 response. Light chains may bind to mast cells, including those in the gut wall, with the rapid release of cytokines (and other mediators). The dual components of this response, by the release of cytokines and other mast cell products, may be the basis for the symptoms associated with cows' milk ingestion.

This research has been co-funded by Food Science Australia and Dairy Australia.

Objectives & Notes

Robert W. Gardner, Ph.D.

Date of talk: Friday, June 10, 2005,
11:30am

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Training:

Current Job Description:	Testing 400 chemicals to determine which “master chemicals” will control their reactions.
Current Faculty Appointments:	Professor Emeritus, Brigham Young University
Medical School/ University Attended	Ph.D. in Animal Science Cornell University, Ithaca, NY
Other Information:	Published <u>CHEMICAL INTOLERANCES: Physiological Causes and Effects and Treatment Modalities</u> CRC Press, 1994 Currently writing a summary of findings as to which master chemicals are effective in controlling reactions to 400 chemicals, and biochemical reasons for their responses. Methodology in testing for sensitivities and monitoring for dosage required to arrest reactions is an important finding in this study.

SPEECH TITLE: “**Master Chemicals Suppressing Multiple Chemical Intolerances**”

At the end of this Presentation, the participant should be able to:

1. Use a tested method of determining chemical sensitivities
2. Learn of dosages of a few “master” chemicals capable of suppressing reactions to over 400 chemicals.
3. Consider the biochemical pathways the “master” chemicals control to terminate reactions.

The above information was provided by the Speaker.

MASTER CHEMICALS SUPPRESSING MULTIPLE CHEMICAL INTOLERANCES

Robert W. Gardner, Ph.D.

Abstract

Chemical intolerances, be they via inhalation or ingestion, confront a large number of helpless patients. Medical professionals are stymied in bringing relief inasmuch as causations and treatments are obscure. An estimated 10-20% of the world population are afflicted with irritable bowel syndrome (1), an example of chemical sensitivities.

My major objective is to share with you my findings relative to possible causes of, a proposed method of testing for, and choices of chemicals in the treatment of chemical intolerances. Motivation for this long time study has been my own total food intolerance, unresolved by a frustrated medical profession. Now constipation has replaced constant diarrhea and flatulence. A pharmacological, rather than immunological, causation has become apparent. I have studied the role of about 400 chemicals in causing reactions, and have identified about a dozen “master” chemicals which suppress reactions to the 400.

Phenolic compounds (Fig. 1, 1A) have surfaced as both causes of, and in certain cases, valuable agents in suppressing reactions. However, other chemicals are also involved in activating reactions and curbing them.

An observation valuable in measuring patient response is changes in pulse. Coca (2) proposed that a change in pulse of 10 beats per minute is indicative of an allergic reaction. The change in pulse is useful as a monitor when using the provocative/neutralization dose test to determine sensitivities, and the neutralizing dose. The neutralization concept was fostered by Clinical Ecologist (now American Academy of Environmental Medicine). Originally food, pollen, etc. extracts were applied sublingually in a 1:10 dilution sequence until the neutralizing dose was found. I used this method to begin. However, Dorothy Sudweeks a chemist assisting Dennis Remington, M.D. while testing patients made a brilliant discovery. She determined that neutralizing doses may be made by preparing a 0.005 M solution of each chemical being tested, and then making 1:1 and 1:9 of the 0.005 M solution (Fig. 2). One drop of the 1:9 dilution applied sublingually will neutralize a reaction if this is proven to be a “control/master” chemical. To test for sensitivity one uses 1 drop of the 1:1 dilution or 0.005M solution applied sublingually, if the patient’s pulse is normal. There is an instantaneous elevation in pulse upon activation of a reaction, or normalization of pulse, when the dosage is the neutralizing dose. An elevation in pulse, or onset of symptoms, verifies a reaction. Then 1 drop of the 1:9 dilution is given, followed by 1 drop of the 1:1 dilution, and finally 3

drops of the 0.005 M solution, with 5-10 seconds between. An additional drop may be required at each step if ethanol was used to increase solubility of chemical, since the volume of the drop decreases. A sequel to this series of drops is required after several applications of the initial dosage; i.e. the neutralizing dose changes upward. Thus 3-4 drops of a 1% solution are needed to neutralize. Eventually 3-4 drops of a 4% solution are needed. I have found 4% to be the upper limit required. At this point chemical powders may be included at 60-200 mg doses in gelatin capsules, to be taken with each meal. Protection lasts 6-7 hours. Incidentally, desensitization is a possibility; more time is needed to determine. These chemicals are not addictive, nor do they cause a "high."

Causes of inflammatory reactions may be attributed to chemicals which trigger the production of prostaglandins, thromboxanes, and leukotrienes (Table 1), as well as activation of voltage sensitive ion channels: Calcium, chloride, potassium, and sodium (3). Prostaglandins, thromboxanes, and leukotrienes are referred to as eicosanoids, which are derived from arachidonic acid (20 carbon unsaturated fatty acids) (4,5). The multiple effects of eicosanoids are summarized in Table 1.

Which chemicals should be used to control reactions inasmuch as one may be reacting to virtually hundreds of chemicals due to chemical intolerances? After hundreds of hours of testing, your speaker has identified "master" chemicals which control reactions to approximately 400 chemicals. Clues came by sublingual challenge with food and pollen extracts, food additives, vitamins, minerals, medications, toxins, etc. A food-chemical dot chart (Fig. 3) illustrates original findings of primarily phenolics assumed to be in foods since the theory then was that one was neutralizing each chemical causing reactions in foods by finding the neutralizing dose of that chemical. Next came a long term study to determine specific chemicals which would block reactions to other chemicals. Blocking the inflammatory cyclooxygenase, lipoxygenase pathways, as well as the calcium, potassium, sodium, and chloride voltage channels, were major objectives. In some cases three or four chemicals could be substituted for each other. Availability, and price, were factors affecting the first choice. Gallic acid proved to be a valuable find since it neutralized reactions to 107 chemicals. Why was it effective? I found it would substitute for verapamil HCl (Fig. 3), an L-type calcium channel blocker (3). Verapamil HCl is now available in extended release tablets. A 180 mg tablet provides protection for a 24 hour period.

Three drops of the 0.005 M solution of gallic acid provides 130 micrograms of the acid, indicative of the minute amounts needed to either block reactions, or activate them. Four drops of a 4% solution of gallic acid amounts to 3.2 mg of the compound. Overdosing with medications is a common problem. For instance, a chemically sensitive patient seeking relief with an initial dose of one or two aspirin tablets at 325 mg each.

Another mistake is for chemically sensitive patients to unwittingly use mega doses of vitamins and /or essential minerals, assuming that this is therapeutic. Surprisingly, they are often part of the problem in these patients.

A second master chemical of note has been the sodium salt of benzene sulfonic acid ($C_6H_5SO_3Na$), effective against 63 diverse chemicals, one being acetylcholine. Indomethacin (relatively selective as a cyclooxygenase 1 inhibitor), substitutes for benzene sulfonic acid, Na salt, suggesting a specific role, since aspirin was not a substitute. Aspirin was useful in countering reactions to 60 chemicals of diverse structures, including α -tocopherol acetate (Vitamin E), hydrogen peroxide, menthol, etc. Quercetin, a flavonoid, is one of the most common phenolic compounds in plants (6). This compound obviously acts as a lipoxygenase blocker since it effectively counters aspirin as a cyclooxygenase inhibitor, in which case arachidonic acid is shunted to the lipoxygenase pathway. Quercetin is also effective against other chemicals, such as norepinephrine, benzene sulfonic acid (Na salt), and is an antihistamine.

Cinnarizine (Fig. 4) is another calcium channel blocker; a central and peripheral vasodilator (3), which I discovered was needed in addition to the calcium channel blockers cited above. It was needed for calcium chloride, carbonate, and gluconate, as well as a diversity of other compounds such as limonene, piperine, etc.

Arginine is the precursor of nitric oxide (7). Amino guanidine (Fig. 3) inhibits both constitutive and inducible nitric oxide synthase (8). It also inhibits the formation of advanced glycosylation end products which have been implicated in the etiology of diabetic complications (8). Sodium nitrate and nitrite are products of catabolism of nitric oxide (7). I learned from testing that amino guanidine likewise suppresses responses to the nitrates, nitrites, L-citrulline, melatonin and other indole ring compounds.

1-Benzylimidazole (Fig. 4) is a thromboxane synthesis inhibitor (9). An important role of this compound is to prevent sneezing if one inhales printer's ink, or other volatile chemicals. In addition, thromboxane A_2 appears to be one of the main mediators of anaphylactically induced longitudinal muscle contractions in the rat small intestine (10).

These chemicals may be used as antidotes for those exposed to toxic substances such as mercury, cyanide, etc.

Vitamin E. (α -tocopherol acetate) is considered to be a biological anti- oxidant (9). In this study it took the role of controlling sodium and chloride channels, and such compounds as phenol, thymol, and thiamine HCl. Ingestion of foods containing thymol leads to stressful abdominal gas production, and repeated belching, in chemically intolerant individuals.

In conclusion, I have not shared all of my findings. I wish to join others of you in verifying these findings, and measure these results in a clinical setting. I am convinced as to the validity of these findings, however, as I have reviewed world literature. Dr. De

Giorgio of Italy (1) recognized our need: “A better understanding of pathogenic mechanisms underlying irritable bowel syndrome may help to develop more effective drugs for this disease.” The pharmaceutical industry could also help the cause by manufacturing more extended release medications.

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Objectives & Notes

Steven Goodman, M.D.

Date of talk: Friday, June 10, 2005, 1:30pm

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Other Information:	Website with full detailed explanation of Intramuscular stimulation: www.northwestims.com

SPEECH TITLE: “**Autonomic Epiphenomena of Segmental Radiculo - Neuropathy**”

At the end of this Presentation, the participant should be able to:

1. Understand regional autonomic dysfunction as the epiphenomena of segmental spinal nerve root pathology and thus the basis for somato-visceral therapy.
2. Recognize & Understand the role of Cannon’s Law of Denervation Supersensitivity in the pathology of regional autonomic dysfunction
3. Understand the clinical manifestations of visceral and somatic autonomic dysfunction supersensitivity and be able to recognize the physical exam findings of the latter.

The above information was provided by the Speaker.

**American Environmental Health Foundation 23rd Annual Symposium:
“The Autonomic Nervous System & Its Relationship to Environmental Pollutants”
Dallas, Texas June 9 - 12, 2005**

Steven R. Goodman, M.D.

Friday June 10, 2005

Autonomic Epiphenomena of Segmental Radiculo-Neuropathy

Introduction

When the equilibrium between ‘Environment’, e.g. Infectious Agents, Chemical & EMF toxicity, etc. and the ‘Self-host’ is disturbed, the ‘Self’ experiences ‘Dis-Ease’. Alternatively, ‘Dis-Ease’ can occur when ‘Self-Defense’ is compromised or weak.

In the first instance, externally applied environmental agents would logically influence the ANS in a manner that depended on the nature of the external agent, its route of administration, distribution and/or predilection for one region or organ-system over another. The spectrum for such exposures would range from ‘entire organism’ in the case of EMF, to only certain organs and tissues for something like a Herpes or Jacob-Creutzfeldt virus. ANS response to such exposures inhabit all points along the analog spectrum from diffuse to regional/localized exist and interact reciprocally, especially the ANS with its Supra-segmental organization. These may be thought of as ‘outside-to-inside’ influences with variable ‘saturation’ and effect on the ANS.

When ‘Self’ is weakened, especially from regional, and in the case of spinal nerve root pathology, segmental pathology, disease may be thought of as spreading from ‘inside-to-out’.

Physical Medicine & Rehabilitation physicians treat patients suffering from a wide variety of disease afflicting the spinal cord and peripheral nerves: multiple trauma and spinal cord victims, as well as Guillain-Barre Syndrome. And of course, common problems like LBP. Therefore our work is pre-occupied with spinal and peripheral nerve pathology.

Peripheral neuropathy, often accompanied by partial denervation, is not exceptional in adults. Of the innumerable causes of nerve damage, such as trauma, metabolic, degenerative, toxic, and other conditions, chronic attrition from spondylosis (the structural disintegration and morphologic alterations that occur in the intervertebral disk, with pathoanatomic changes in surrounding structures) is by far the most common.

The spinal nerve root, because of its vulnerable position, is notably prone to injury from pressure, stretch, angulation, and friction. The spinal nerve root (s.n.r.) is especially vulnerable to micro & macro-trauma because of its localized anatomy consisting of tethering by the dura mater and spinal ligaments. Other causes of radiculopathy (i.e., neuropathy at the nerve root), such as arachnoiditis, neuroma, and intraspinal tumors, are much less common. Spondylosis increases with age; therefore, spondylosis pain is more common in middle aged individuals who have accumulated an injury pool, an accumulation of repeated major and minor injuries to a segment leading to unresolved clinical residuals that may, or may not, produce pain. The s.n.r. consists of 3 primary divisions: motor, sensory, and autonomic fibres. The sensory dorsal root ganglion occupies a large proportion of the s.n.r. cross-sectionally.

Cannon and Rosenblueth's Law of Denervation

This law is seldom cited to explain neuropathic pain; it deserves to be better known. It points out that the normal physiology and integrity of all innervated structures are dependent on the arrival of nerve impulses via the intact nerve to provide a regulatory or *trophic* effect. When this flow, which is probably a combination of axoplasmic flow and electrical input, is blocked, innervated structures are deprived of the trophic factor, which is vital for the control and maintenance of cellular function.

A-trophic structures become highly irritable and develop abnormal sensitivity or supersensitivity according to Cannon and Rosenblueth's Law of Denervation (7): "When a unit is destroyed in a series of efferent neurons, an increased irritability to chemical agents develops in the isolated structure or structures, the effect being maximal in the part directly denervated."

All denervated structures develop supersensitivity (including skeletal muscle, smooth muscle, spinal neurons, sympathetic ganglia, adrenal glands, sweat glands, and brain cells). Cannon and Rosenblueth's original work was based on total denervation or decentralization for supersensitivity to develop; accordingly, they named the phenomenon denervation supersensitivity. **But it is now known that physical**

interruption and total denervation are not necessary: Any circumstance that impedes the flow of motor impulses for a period of time can rob the effector organ of its excitatory input and cause disuse supersensitivity in that organ and, significantly, in associated spinal reflexes.

The importance of disuse supersensitivity cannot be over emphasized. When a nerve malfunctions, the structures it supplies become supersensitive and behave abnormally. These structures overreact to many forms of input, not only chemical, but physical inputs as well, including stretch and pressure.

Supersensitive muscle cells can generate spontaneous electrical impulses that trigger false pain signals or provoke involuntary muscle activity (9). Supersensitive nerve fibers become receptive to chemical transmitters at every point along their length instead of at their terminals only. Sprouting may occur, and denervated nerves are prone to accept contacts from other types of nerves including autonomic and sensory nerve fibers (10). **Short circuits are possible between sensory and autonomic (vasomotor) nerves and may contribute to complex regional pain syndrome.**

Disuse supersensitivity is basic and universal, yet not at all well known or credited- The important role of supersensitive structures after neuropathy or denervation was previously neglected. Many diverse pain syndromes of apparently unknown causation may be attributed to the development of hypersensitive receptor organs and supersensitivity in pain sensory pathways. Instead of nociception, there can be severe pain in response to a noxious stimulus (hyperalgesia) or severe pain in response to a stimulus that is not normally noxious (allodynia).

Disuse supersensitivity affects sympathetic ganglia and the smooth muscles of the GI, respiratory, vascular, genitor-urinary systems. Smooth muscle cells become supersensitive to Ach (acetylholine) requiring a lower voltage for contraction > gap formation > leakage > **trophic edema**. Visceral and Somatic (muscle) organ systems affected.

Somatic (musculoskeletal)

- **Vasoconstriction**-cooling
- **Pilomotor** reflexes are hyperactive: gooseflesh
- **Sudomotor** reflexes are hyperactive: hyperhidrosis

Visceral: 'functional' disturbances of smooth muscle lining hollow-organs:

- bronchioles: asthma
- mucous membranes: sinusitis
- Gastrointestinal: IBS
- genito-urinary: infertility, prostatitis, irritable bladder
- vascular: cardiac angina, renal-vascular disease

Intramuscular Stimulation Rx as Somato-Visceral Therapy for ANS Dysfunction

Physical Therapy and Stimulation-Induced Analgesia

Melzack & Wall's Gate Theory articulates the empirical observation that stimulation of fast conducting large diameter fibres from mechanoreceptors, as when a person rubs quickly the skin over a painful soft-tissue to induce temporary analgesia. Of all large-diameter fast-conducting fibers, the 1A afferent muscle proprioceptors constitute the largest diameter ones. Sherrington estimated that 40% of muscle afferents are proprioceptors. Therefore it appears reasonable to suspect that dysfxn in this receptor-fiber type could play an important role in the modulation of pain, and the ANS as well.

Physical therapy is widely used as a first-line treatment for peripheral neuropathic pain. Early physical treatment is advocated, because earlier treatment is said to correlate with better outcome. Neuropathic pain is a hypersensitivity phenomenon, and its treatment requires desensitization. Lomo has shown in animal experiments that hypersensitivity and other features of denervated muscle can be reversed by electric stimulation

Physical therapy also achieves its effect by stimulation. Local therapy excites receptors (in skin and muscle); for example, massage activates tactile and pressure receptors; exercise, manipulation, and dry needling stimulate muscle spindles and Golgi organs; heat and cold act on thermal receptors. These stimuli are sensed by their specific receptors, transduced into nerve impulses, and relayed to the dorsal horn. All forms of physical therapy, including dry needling, are effective only when the nerve to the painful part is still intact. A dry needling technique called intramuscular stimulation is often effective. In intramuscular stimulation, diagnosis, treatment, as well as progress during therapy are determined according to physical signs of neuropathy. The effective application of intramuscular stimulation therefore requires a sound background both in anatomy and neurophysiology (27).

Stimulation also can be applied directly to the spinal cord (28) Cut and colleagues have reported that neuropathic pain may be effectively relieved by electric stimulation of the

spinal cord. Stimulation of the spinal cord has been shown to normalize withdrawal response thresholds in a rat model. The effect of stimulation of the spinal cord on neuropathic pain and allodynia is believed to be caused by inhibition of glutamate and aspartate release at NMDA receptor sites, and activation of local GABAergic mechanisms.

Conclusions:

- 1) Spinal nerve root pathology is the most common form of neuropathy: spondylosis is 'universal'.
- 2) S.N.R. pathology will manifest variable degrees of motor, sensory and/or autonomic fiber involvement; based on fiber size, large muscle afferents and motor neurons figure prominently; autonomic involvement not generally as well appreciated!
- 3) Cannon & Rosenbleuth's Law of Denervation Supersensitivity: now Disuse/Dysfunction Supersensitivity: somatic and visceral end-organs are targets; increased skeletal & smooth muscle irritability/spasm, edema, pain
- 4) Supersensitivity (pain and ANS hyper-reactivity) can be reversed through Counter-Reflex Stimulation Induced Analgesia and **NORMO**-sensitization (vs. **DE**-sensitization).
- 5) Skeletal muscle proprioceptive fibers constitute a significant portion of afferent sensory fibers and so a potentially powerful point of afferent stimulation.
- 6) Intramuscular Stimulation is a unique therapy that takes advantage of muscle proprioceptive input to stimulate spinal reflexes and reverse supersensitivity syndromes.
- 7) Segmental supply of ANS directs which myotomes to treat with IMS to reverse 'regional' ANS syndromes.

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Objectives & Notes

Dietrich K. Klinghardt, M.D., Ph.D. Date of talk: Friday, June 10, 2005, 2:00pm

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Board Certifications:	Board certified in General Practice (Germany) and Pain Management, US

SPEECH TITLE: “Toxicity of Autonomic Ganglia and Plexi Diagnosis and Treatment”

At the end of this Presentation, the participant should be able to:

1. Be aware of the many regulatory functions the facial and cervical autonomic ganglia have.
2. Be knowledgeable of the different classes of neurotoxins that have high affinity for these ganglia.
3. Understand specific and targeted detoxification strategies.

The above information was provided by the Speaker.

Toxicity of Autonomic Ganglia and Plexi: Diagnosis and Treatment

The ganglia of the ANS can be considered brains outside the brain: they produce well over 70 neuropeptides which are involved in most of our physiological processes, such as the dilation and contraction of blood vessels, the activation and silencing of lymphocytes and other white cells, the metabolic activity of the liver cells, etc. Many of the post-ganglionic ANS-projections lead to a presynaptic bulb in the endothelium of the blood vessels. The receiving post-synaptic bulb is either on a white blood cell, a brain cell or elsewhere. Depending on the composition of the neuropeptides squirted into the blood stream the ganglia regulate and influence our immune-system and our emotions. Neurotoxins can damage, up- or down- regulate affected ganglia and cause severe disturbance in the dysregulated system. We commonly find one of four distinct groups of neurotoxins involved in ganglionic toxicity and dysregulation:

1. heavy metals, 2. mycotoxins, 3. bacterial exo- and endotoxins, 4. xenobiotics

Only for principal diagnostic non-invasive techniques are available to diagnose toxicity of the ANS ganglia:

1. R. Voll's electroacupuncture (electro-dermal screening or EDS), 2. clinical observation (and therapeutic trial), 3. autonomic response testing, 4. functional MRI.

The following treatments are available and effective:

1. acupuncture and its offshoots (auricular medicine, trigger point injections, etc.), 2. ganglion injections (neural therapy and related techniques), 3. transdermal treatment: iontophoresis, laser photopheresis, phonopheresis, essential oils, 4. osteopathic manual treatment ("ANS manipulation") Oral treatment of selected ganglia has been attempted (i.e. Viagra) but is non-specific and non-lasting. The correct diagnosis and resulting specific treatment of injured ganglia can be most rewarding leading to excellent results in such varied conditions as chronic pain syndromes, impotence, MCS, tinnitus, migraine and sinusitis, glaucoma, angina, seizures and many others.

Objectives & Notes

John Laseter, Ph.D.

Date of talk: Friday, June 10, 2005, 2:30pm

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Other Information:	Over 105 publications in peer-reviewed journals and books (1966-2001)

SPEECH TITLE: “Polybrominated Diphenyl Ether and Human Health”

At the end of this Presentation, the participant should be able to:

1. Recognize the chemical and physical nature of PBDE's
2. Understand their origin, distribution and potential health effects.
3. Other chemicals of similar structure will be discussed in terms of human health effects.

The above information was provided by the Speaker.

Polybrominated Diphenyl Ethers, Triclosan and Perchlorates and Their Impact on Human Health”

John L. Laseter, Ph.D.
Accu-Chem Laboratories
Richardson, TX

The objectives of this presentation will be to introduce several classes of pollutants that are entering the environment at a previously unrecognized rate. Many of these chemicals are being observed in mother's milk, fat, blood and other body fluids and tissues. Although it is not clear what the health effects are, it is known that the poly brominated diphenyls ethers (PBDE's) and perchlorated will impact on the adult thyroid and can result in neurological damage in infants. Also, some anti-bacterial agents are now becoming the top organic pollutants in U.S. rivers and lakes. However, health effects of the daily intake of these latter compounds are poorly understood.

Better monitoring of both the environment and humans will be required in the future to gain an understanding of that relationship in cases where exposures have occurred to pregnant women. It may be prudent to establish the levels in milk to understand exposure to the infant at postpartum.

Objectives & Notes

Lucie Lessard, M.D.

Date of talk: Friday, June 10, 2005, 3:30pm

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Other Information:	Publications in Plastic Surgery – Head and Neck Cancer and Craniofacial distraction Osteogenesis (Basic Sciences)

SPEECH TITLE: “Autonomic Disorders with Breast Implant Reconstruction”

At the end of this Presentation, the participant should be able to:

1. to understand better the association of some autonomic disorders with permanent foreign body materials like breast prosthesis.
2. to have a good knowledge of what is in the literature in relationship with breast prosthesis and clinical symptomatology.
3. to acquire the concepts of treatment when these symptoms occurred and the outcome.

The above information was provided by the Speaker.

Abstract

Title: Autonomic Disorders (Drop Attacks) in Breast Implant Reconstructed Patients

The medical implication of the presence of Breast Implants is still not clear scientifically. After the silicone implants moratorium in 1992, many studies were done and are now important in the surgical community for the benefit of the patient. Breast implants are used for esthetic reasons but are more valuable post-breast cancer patient in Reconstructive surgery when a TRAM flap is not indicated.

We are presenting 2 cases of Breast Implant used for reconstructive purposes who presented to us many years after their initial surgery with severe drop attacks and other autonomic disorders. The investigation of the first case is presented in details and a testimony from the patient is included in a short video clip.

The patients were eventually treated surgically when the complete neurological investigation was found to be negative for a specific pathology. The implants were removed and the drop attacks and other Raynaud's like phenomenon reversed immediately post-operatively faster than we expected.

A review of the literature did not reveal any other reported cases of drop attacks associated to Breast Implants. Specific review of autonomic disorders and breast implants revealed no item found. Some of the few key articles concerning breast implants and some systemic problems are also reviewed.

References:

1. Gabriel SE et al: Risk of connective-tissue diseases and other disorders after breast implantation N Engl J Med 330 (24):1697, June 16, 1994
2. Sanchez-Guerrero J, Colditz GA, Karlson EW, et al: Silicone breast implants and the risk of connective-tissue diseases and symptoms. N Engl J Med 332(25):1666, June 22, 1995
3. Birdsell DC, Jenkins H, Berkel H: Breast cancer diagnosis and survival in woman with and without breast implants Plast Reconstr Surg 92:795,1993
4. Peters W et al: An outcome analysis of 100 women after explantation of silicone gel breast implants – Ann Plast Surgery 1997, Jul: 39 (1) 9-19

Objectives & Notes

Steven Goodman, M.D.

Date of talk: Friday, June 10, 2005, 4:00pm

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Training:

Current Job Description:	Private Practice Physical Medicine & Rehabilitation
Current Faculty Appointments:	(Previous/not currently) University of Washington – Multidisciplinary Pain Center
Medical School/University Attended:	S.U.N.Y and Buffalo
Internship:	UCSF/Mt. Zion Hospital & Medical Center San Francisco
Residency:	University of Washington
Board Certifications:	American Board of Physical Medicine & Rehabilitation
Other Information:	Website with full detailed explanation of Intramuscular stimulation: www.northwestims.com

SPEECH TITLE: **“Intramuscular Stimulation Therapy for Autonomic Epiphenomena of Segmental Radiculo-Neuropathy”**

At the end of this Presentation, the participant should be able to:

4. Understand intramuscular stimulation as a form of segmented counter-irritation reflex stimulation that reverses dysfunction supersensitivity.
5. Understand the role of the acupuncture needle in eliciting spiral reflexes, creating a ‘current of injury’, and reversing muscle contractures and autonomic dysfunction
6. Examine a patient with autonomic dysfunction and identify the appropriate anatomical levels and locations for targeting intramuscular stimulation.

The above information was provided by the Speaker.

**American Environmental Health Foundation 23rd Annual Symposium:
“The Autonomic Nervous System & Its Relationship to Environmental Pollutants”
Dallas, Texas June 9 - 12, 2005**

Steven R. Goodman, M.D.

Friday June 10, 2005

**Intramuscular Stimulation Therapy for Autonomic Epiphenomena of Segmental
Radiculo-Neuropathy**

Introduction

RADICULOPATHY: ITS FREQUENT RELATIONSHIP TO SPONDYLOSIS

It is not unusual for the flow of nerve impulses to be obstructed; peripheral neuropathy, often accompanied by partial denervation, is not exceptional in adults. Of the innumerable causes of nerve damage, such as trauma, metabolic, degenerative, toxic, and other conditions, chronic attrition from spondylosis (the structural disintegration and morphologic alterations that occur in the intervertebral disk, with pathoanatomic changes in surrounding structures) is by far the most common.

The spinal nerve root, because of its vulnerable position, is notably prone to injury from pressure, stretch, angulation, and friction. Other causes of radiculopathy (i.e., neuropathy at the nerve root), such as arachnoiditis, neuroma, and intraspinal tumors, are much less common. Spondylosis increases with age; therefore, spondylotic pain is more common in middle aged individuals who have accumulated an *injury pool*, an accumulation of repeated major and minor injuries to a segment leading to unresolved clinical residuals that may, or may not, produce pain (11).

Ordinarily, spondylosis follows a gradual, relapsing, and remitting course that is silent, unless and until, symptoms are precipitated by an incident often so minor that it passes unnoticed by the patient. All gradations of spondylosis can exist, but early or incipient spondylotic changes, even when unsuspected, can nevertheless irritate and upset function in the segmental nerve.

The emphasis on radiculopathy is not without reason: With an acute injury to a healthy nerve, there is no prolonged discharge of pain signals, whereas the same injury to a

neuropathic nerve can cause a sustained discharge (12). In other words, for pain to become a persistent symptom, the affected fibers must be previously irritated or defective. That is why some people develop severe pain after an apparently minor injury, and why that pain can continue beyond a reasonable period.

The manifestations of neuropathic dysfunction are motor, sensory, and autonomic. In our studies, early and subtle signs of peripheral neuropathy were found in a significant number of young (under 30 years), apparently normal, and asymptomatic individuals (13). Brief and transient motor manifestations are the first to appear, and radiculopathy can occur without pain. Muscle shortening is an early and regular feature of radiculopathy, because large diameter nerve fibers at the nerve root-axons of motoneurons and myelinated primary afferents (muscle proprioceptors)-are the first to suffer physically. Painless, reversible, tight muscle knots can be felt in most individuals; not uncommonly, even in toddlers. Pain is not therefore a feature of radiculopathy unless nociceptive pathways are involved. Many neuropathies are pain free, such as sudomotor hyperactivity in hyperhidrosis, and muscle weakness in ventral root disease.

Cannon and Rosenblueth's Law of Denervation

This law is seldom cited to explain neuropathic pain; it deserves to be better known. It points out that the normal physiology and integrity of all innervated structures are dependent on the arrival of nerve impulses via the intact nerve to provide a regulatory or *trophic* effect. When this flow, which is probably a combination of axoplasmic flow and electrical input, is blocked, innervated structures are deprived of the trophic factor, which is vital for the control and maintenance of cellular function.

A-trophic structures become highly irritable and develop abnormal sensitivity or supersensitivity according to Cannon and Rosenblueth's Law of Denervation (7): "When a unit is destroyed in a series of efferent neurons, an increased irritability to chemical agents develops in the isolated structure or structures, the effect being maximal in the part directly denervated."

All denervated structures develop supersensitivity (including skeletal muscle, smooth muscle, spinal neurons, sympathetic ganglia, adrenal glands, sweat glands, and brain cells). Cannon and Rosenblueth's original work was based on total denervation or decentralization for supersensitivity to develop; accordingly, they named the

phenomenon denervation supersensitivity. But it is now known that physical interruption and total denervation are not necessary: Any circumstance that impedes the flow of motor impulses for a period of time can rob the effector organ of its excitatory input and cause disuse supersensitivity in that organ and, significantly, in associated spinal reflexes (8).

The importance of disuse supersensitivity cannot be over emphasized. When a nerve malfunctions, the structures it supplies become supersensitive and behave abnormally. These structures overreact to many forms of input, not only chemical, but physical inputs as well, including stretch and pressure.

Supersensitive muscle cells can generate spontaneous electrical impulses that trigger false pain signals or provoke involuntary muscle activity (9). Supersensitive nerve fibers become receptive to chemical transmitters at every point along their length instead of at their terminals only. Sprouting may occur, and denervated nerves are prone to accept contacts from other types of nerves including autonomic and sensory nerve fibers (10). Short circuits are possible between sensory and autonomic (vasomotor) nerves and may contribute to complex regional pain syndrome.

Disuse supersensitivity is basic and universal, yet not at all well known or credited- The important role of supersensitive structures after neuropathy or denervation was previously neglected. Many diverse pain syndromes of apparently unknown causation may be attributed to the development of hypersensitive receptor organs and supersensitivity in pain sensory pathways. Instead of nociception, there can be severe pain in response to a noxious stimulus (hyperalgesia) or severe pain in response to a stimulus that is not normally noxious (allodynia).

Diagnosis

Diagnosing pain and dysfunction caused by radiculopathy depends almost entirely on the examiner's clinical experience and acumen. The history gives little assistance. Pain often arises spontaneously with no history of trauma, or else the degree of reported pain far exceeds that consistent with the injury. Laboratory and radiologic investigations are generally not helpful. Thermography reveals decreased skin temperature in affected dermatomes and this can be an indication of neuropathy, but does not necessarily signify pain.

Radiculopathies are difficult to document with routine nerve conduction studies, which measure only the few fastest conducting and largest fibers and take no account of the

majority of smaller fibers. In focal neuropathy, nerve conduction velocities remain within the wide range of normal values, but F-wave latency may be prolonged. Electromyography is not specific either.

The physical signs of neuropathy are distinctive and different from the well-known ones of outright denervation, such as loss of sensation and reflexes. They are important to look for because they indicate early neural dysfunction for which no satisfactory laboratory or imaging test exists. A careful inspection for signs of motor, sensory, and autonomic (vasomotor, sudomotor, and pilomotor) dysfunction in the skin and affected muscles is necessary. Vasoconstriction differentiates neuropathic pain from inflammatory pain: In neuropathic pain, affected parts are perceptibly colder. There may be increased sudomotor activity and the pilomotor reflex is often hyperactive and visible in affected dermatomes as goose bumps (Fig. 28-1). There can be interaction between pain and autonomic phenomena. A stimulus such as chilling, which excites the pilomotor response, can precipitate pain; vice versa, pressure on a tender motor point can provoke the pilomotor and sudomotor reflexes.

Increased permeability in blood vessels can lead to local subcutaneous tissue edema (neurogenic edema or trophedema). This can be seen as *peau d'orange* skin (Fig. 28-2) and confirmed by the match stick test. Trophedema is non-pitting to digital pressure, but when a blunt instrument such as the end of a match stick is used, the indentation produced is clear-cut and persists for many minutes (Fig. 28-3). This quick and simple test can demonstrate neuropathy earlier than electromyography. Trophic changes such as dermatomal hair loss may also accompany neuropathy.

Knowledge of the Segmental Nerve Supply to Muscles Is a Clue to Diagnosis

Neuropathic changes are primarily in muscle. Even when symptoms appear to be in joints or tendons, signs in the muscles are the most consistent and relevant: increased muscle tone; tenderness over motor points; and taut and tender, palpable contracture bands and restricted joint range. Each constituent muscle must be palpated and its condition noted. Palpation requires detailed knowledge of anatomy, and clinical skill comes only with practice. Moreover, because many paraspinal muscles are compound (e.g., the longissimus) and extend throughout most of the length of the vertebral column, the entire spine must be examined even when symptoms are localized to one region.

Physical Therapy and Stimulation-Induced Analgesia

Physical therapy is widely used as a first-line treatment for peripheral neuropathic pain. Early physical treatment is advocated, because earlier treatment is said to correlate with better outcome. Neuropathic pain is a supersensitivity phenomenon, and its treatment requires desensitization. Lomo has shown in animal experiments that supersensitivity and other features of denervated muscle can be reversed by electric stimulation (26).

Physical therapy also achieves its effect by stimulation. Local therapy excites receptors (in skin and muscle); for example, massage activates tactile and pressure receptors; exercise, manipulation, and dry needling stimulate muscle spindles and Golgi organs; heat and cold act on thermal receptors. These stimuli are sensed by their specific receptors, transduced into nerve impulses, and relayed to the dorsal horn. All forms of physical therapy, including dry needling, are effective only when the nerve to the painful part is still intact. A dry needling technique called intramuscular stimulation (see Chapter XX) is often effective. [Ed/AU: 5] In intramuscular stimulation, diagnosis, treatment, as well as progress during therapy are determined according to physical signs of neuropathy. The effective application of intramuscular stimulation therefore requires a sound background both in anatomy and neurophysiology (27).

Chronic myofascial pain is not ordinary nociception: Deqi pain sensations are not normal because they are associated with receptors that sense muscle shortening (proprioceptors). The classic acupuncturist demonstrates this by the needle grasp occurring at the site of penetration when a neuropathic muscle is needled. Needling is usually pain free when an acupuncture needle enters a normal muscle, but when the needle pierces a shortened muscle, it produces a cramp, and the needle is observed to be firmly grasped by the shortened muscle. The intensity of the needle grasp parallels the degree of muscle shortening, and it gradually eases off during treatment as muscle shortening is released: Release frequently occurring in minutes. Because muscle pain eases concurrently with the release of the needle grasp, patients soon become aware of the importance of eliciting the Deqi sensation and releasing needle grasp during treatment.

Conclusions:

- 1) Intramuscular Stimulation is a form of Counter-Irritation Reflex Stimulation that reverses Disuse/Dysfunction Supersensitivity, including skeletal muscle contractures and ANS Dysfunction by initially stimulating muscle proprioceptors.

- 2) The 'Needle Effect' has additional unique therapeutic results; IMS induces a 'Current of Injury' that stimulates healing and helps to normalize the electrical state of the neuromuscular apparatus.
- 3) Proper physical examination and diagnosis of Radiculo-Neuropathic Pain & Autonomic Syndromes is critical to identification of the appropriate somatic levels to direct IMS Rx for ANS Dysfunction.
- 4) PAIN is actually of 3 distinct functional types, and the IMS acupuncture needle induces a particular type of pain classically referred to as 'Deqi Response' by acupuncturists.
- 5) Reduction of ANS mediated symptoms will follow release of appropriate segmental skeletal muscle contractures through somato-visceral reflexes.

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Objectives & Notes

Mohamed B. Abou-Donia, Ph.D. Date of talk: Friday, June 10, 2005, 4:30pm

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Medical School/ University Attended	University of California, Berkeley
Board Certifications:	American Board of Toxicology (ABT), and Academy of Toxicological Sciences (ATS)
Other Information:	Book Editor, <i>Neurotoxicology</i> , CRC; Publications: more than 300.

SPEECH TITLE: **“Chemical-Induced Developmental Neurotoxicity”**

At the end of this Presentation, the participant should be able to:

1. Appreciate that chemicals are transported during gestation from the mother across the placental to the fetus.
2. Know that gestational exposure to chemicals causes neurotoxicity manifested as neurological deficits.
3. Understand that chemical-induced neurotoxicity is characterized by neurobehavioral and brain cell death.

The above information was provided by the Speaker.

Chemical-Induced Developmental Neurotoxicity.

Mohamed B. Abou-Donia, Wasi A. Khan, Anjelika M. Dechkovskaia, Larry B. Goldstein, Sarah L. Bullman, and Ali A. Abdel-Rahman Department of Pharmacology and Cancer Biology, and Department of Medicine, Duke University Medical Center, Durham, North Carolina

This study was carried out to investigate interactive neurotoxic effects of combined exposure, during gestation to real-life levels of the cholinotoxicants, nicotine, and the organophosphorus insecticide, chlorpyrifos on postnatal development of the cholinergic system and its long-term functional consequences in the rat. Nicotine is a direct cholinergic agonist at the nicotinic acetylcholine receptors, delivered by cigarette smoking, and chlorpyrifos is an inhibitor of acetylcholinesterase (AChE), resulting in accumulation of acetylcholine at the muscarinic and nicotinic receptors and their subsequent over-stimulation. Groups of 5-timed pregnant Sprague-Dawley rats were treated daily with nicotine (1mg/kg, sc), chlorpyrifos (0.1 mg/kg, dermal), or combination of both on gestational days (GD) 4-20. On postnatal day (PND) 7, 30, and 60 there was a significant increase in AChE activity in some brain regions caused by all treatments. Increased AChE activity, decreases acetylcholine (ACh) required for cholinergic neurotransmission, and slows down cholinergic functions. At PND 30 and 60, male offspring from gestational treatment with nicotine and female offspring from gestational treatment with nicotine and nicotine/chlorpyrifos showed increased glial fibrillary acidic protein (GFAP) immunostaining in the CA1 subfield of the hippocampus and/or cerebellum. Increased GFAP expression is an indication of neuronal injury. The same treatment groups also exhibited neuronal Purkinje cell loss at the two-time points. Other groups of pregnant rats were treated with a daily dose of 3.3 mg/kg nicotine via an implanted mini osmotic pump, 1.0 mg/kg chlorpyrifos, dermal, or both. On PND 90,

offspring from all treatment groups, showed elevated AChE activity in some brain regions and significant Purkinje cell loss and increased GFAP in the granular cell layer and the white matter in the cerebellum. At this time-point, animals from all treatments exhibited sensorimotor deficits. The results also indicated that combined exposure to both chemicals was more effective in causing neuronal injury than each chemical alone. Collectively, gestational exposure to real-life, low levels of nicotine, chlorpyrifos, alone or in combination caused neurochemical, neuropathological, and neurobehavioral deficits, in the offspring later in life. By inference, exposure of humans to these chemicals during gestation, can lead to neurological disorders during childhood, adolescence, and adulthood.

(Supported in part by a U.S. EPA grant number R829399-01-0).

Objectives & Notes

Allan D. Lieberman, M.D.

Date of talk: Friday, June 10, 2005,
5:00pm

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Board Certifications:	American Board Env. Medicine

CASE TITLE: “The Dysautonomic Pattern of Environmentally Triggered Disease”

At the end of this Presentation, the participant should be able to:

1. Recognize the multisystemic pattern of environmentally triggered disease and its mechanism.
- 2.

3.

The above information was provided by the Speaker.

**THE AUTONOMIC NERVOUS SYSTEM MANIFESTATIONS OF
ENVIRONMENTALLY TRIGGERED DISEASE
PRESENTATION OF A NEW HYPOTHESIS**

Twenty- five years of practicing Environmental Medicine involving over 11,000 patients led me to consider other mechanisms of injury to explain the phenomenon we call chemical sensitivity. Chemical sensitivity is an acquired disorder and in the two available studies almost all the affected patients could tell you the specific events that precipitated the onset of their illness. Pesticides were clearly a major cause and although chemical sensitivity is a multi-system disorder, the nervous system seems to take the greatest hit.

These patients have almost total awareness of their body in contrast to the so called normal person whose sensory input to the brain is nearly totally inhibited, thus achieving what the Indian yogas sought to achieve – a loss of awareness of ones self. We are taught that God created the autonomic nervous system because he knew that if man were to be in charge of his visceral functions, he would surely mess it up. This is, in my opinion, what happens to the chemically sensitive patient and brings us to the central theme of this meeting: The role of the autonomic nervous system in environmentally triggered disease.

I hypothesize that there can be no better model for chemical sensitivity than the person poisoned by organophosphate pesticide. Acute poisoning is very predictable as inhibition of acetylcholine esterase results in the build up of acetylcholine resulting in muscarinic, nicotinic and central nervous system effects.

**SIGNS AND SYMPTOMS OF ANTI-CHOLINESTERASE INSECTICIDE
POISONING**

NERVOUS TISSUE AND RECEPTORS AFFECTED	SITE AFFECTED	MANIFESTATIONS
Parasympathetic autonomic postganglionic nerve fibers (muscarinic receptors)	Exocrine glands	Increased salivation, lacrimation, perspiration
	Eyes	Miosis (pinpoint and non-reactive), ptosis, blurring of vision, conjunctival injection, "bloody tears"
	Gastrointestinal tract	Nausea, vomiting, abdominal tightness, swelling and cramps, diarrhea, tenesmus, fecal incontinence
	Respiratory tract	Excessive bronchial secretions, rhinorrhea, wheezing, edema, tightness in chest, bronchospasms, bronchoconstriction, cough, bradypnea, dyspnea
	Cardiovascular system	Bradycardia, decrease in blood pressure
	Bladder	Urinary frequency, incontinence
Parasympathetic and sympathetic autonomic fibers (nicotinic receptors)	Cardiovascular system	Tachycardia, pallor, increase in blood pressure
Somatic motor nerve fibers (nicotinic receptors)	Skeletal muscles	Muscle fasciculations (eyelids, fine facial muscles), cramps, diminished tendon reflexes, generalized muscle weakness in peripheral and respiratory muscles, paralysis: flaccid or rigid tone Restlessness, generalized motor activity, reaction to acoustic stimuli, tremulousness, emotional lability, ataxia
Brain (acetylcholine receptors)	Central nervous system	Drowsiness, lethargy, fatigue, mental confusion, inability to concentrate, headache, pressure in head, generalized weakness Coma with absence of reflexes, tremors, Cheyne-Stokes respiration, dyspnea, convulsions, depression of respiratory centers, cyanosis

Reference: Ecobichon DJ, Joy RM. Pesticides and neurological diseases, 2nd ed. Boca Raton, FL, CRC Press, 1993, 230.

These signs and symptoms simulate the clinical presentation reported by the chemically sensitive patient.

But esterases have other functions beside the break down of acetylcholine. They have a major role in lymphocyte function via serine hydrolase an esterase. This ties in the immune system to my hypothetical model and explains the altered resistance to infection and neoplasia suffered by organophosphate pesticide exposed people.

FUNDAMENTAL AND APPLIED TOXICOLOGY 16, 61-74 (1991)

Modulation of Interleukin-2 Driven Proliferation of Human Large Granular Lymphocytes by Carbaryl, an Anticholinesterase Insecticide

SINA BAVARI, GEORGE P. CASALE, ROGER E. GOLD, AND EDWARD F. VITZTHUM

“The immunotoxic consequences of occupational and other environmental exposures to carbaryl and other anticholinesteratic insecticides are not yet defined; however, it is significant that carbaryl targeted diverse IL2-dependent immunologic processes at concentrations that produce little or no inhibition of serum cholinesterase...”

Esterases play an important role in metabolizing a whole host of aromatic and aliphatic hydrocarbons and participate in the break down of the organophosphate pesticide itself as well as the break down of the second most common group of pesticides – the pyrethroids.

Pesticide manufactures label their products with the warning:

It was after seeing these warnings that I began developing the hypothesis that chemical sensitivity was related to our universal exposures to esterase inhibitors and the consequences of these exposures. It would explain why our patients react to so many places and things, as there is virtually no place on the planet where esterase-inhibiting chemicals are not present. But there is another explanation. Critical to this hypothesis is the research of Duffy, Burchfield and Ecobichon who demonstrated that:

The initial elevated levels of acetylcholine causes the postsynaptic receptor to become more sensitive to endogenous neurotransmitter and as the patient is continually producing acetylcholine its build up or the body's heightened response to the neurotransmitter results in chronic ever ongoing muscarinic, nicotinic and central nervous system effects. Proof of this is that treatment of chemically sensitive patients with small doses of atropine, the specific antidote to organophosphate pesticides, is often very effective. The smallest dose being 0.2 mg.

To argue this case further we must recognize that there are four separate clinical manifestations of organophosphate pesticide poisoning.

Organophosphate Pesticide Poisoning and Patterns of Injury

manifestations

all independent of cholinergic

properties

relating to their anti-AchE

Peripheral Autonomic CNS

Anti-cholinesterase Effect

not related to

AchE or NTE

What is important to recognize is that the intermediate syndrome, the delayed neuropathy and the chronic induced neuropsychiatric disorder can occur without an overt organophosphate pesticide reaction. The patient may never know that he or she was exposed. To amplify the significance of this I would like to quote from the Lancet Editorial of August 15, 1998.

THE LANCET

Organophosphorus compounds: good, bad, and difficult

“...the difficult issue about the safety of organophosphorus agents is not acute safety but whether chronic low-dose exposure to organophosphorus compounds causes neuropsychiatric disease without producing acute cholinergic symptoms.”

You cannot protect yourself from something that you do not know that you have been exposed.

To sum up my presentation, I have often conceived of chemical sensitivity as a dysautonomic or an overt autonomic nervous system disorder acquired when chance brings genetically marked individuals in contact with a toxic chemical exposure.

References:

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Bavari S, Casale G, Gold R, Vitzthum E. Modulation of interleukin-2-driven proliferation of human large granular lymphocytes by carbaryl, an anticholinesterase insecticide. *Fund Appl Toxicol* 1991;17:61-74.

**23rd Annual International Symposium
on
Man and His Environment in Health and Disease**

**Special Focus
The Autonomic Nervous System and Its Relationship to Environmental
Pollutants Including the Cardiovascular System and Electromagnetic
Sensitivity**

**Sponsored by
American Environmental Health Foundation
and
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Physician Accreditation/Credit:

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education and the American Environmental Health Foundation. The University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education is accredited by the ACCME to provide continuing medical education for physicians.

The University of North Texas Health Science Center at Fort Worth is accredited by the American Osteopathic Association to award continuing medical education to physicians.

The University of North Texas Health Science Center at Fort Worth designates this educational activity for a maximum of 24 Category 1 credits toward the AMA Physician's Recognition Award. Each physician should claim only those credits that he/she actually spent in the activity.

The University of North Texas Health Science Center anticipates this program for 24 hours in Category 2A CME credit hours, pending approval from the American Osteopathic Association.

Nursing Accreditation/Credit:

University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education, Provider #02588A, is approved provider of continuing nursing education by the Texas Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation. This activity

meets Type 1 criteria for mandatory continuing education requirements toward relicensure as established by the Board of Nurse Examiners for the State of Texas. This activity is approved for 28.8 Contact Hours.

To receive a certificate of successful completion, participants must attend the activity in its entirety and complete and return the activity evaluation credit request form.

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FINANCIAL CONSIDERATION

AEHF is a nonprofit organization that was founded in 1975 to provide education and research into Environmental Medicine. This year's Symposium is our 23rd Annual International Symposium and is our major vehicle for educating the medical professional.

Funding for the symposium is provided by registration fees from physicians and exhibitors. Proceeds from the AEHF store cover the shortfall between registration fees and expenses for the conference. AEHF does not receive grants or any outside financial support for our education. Donations are accepted and used toward research into environmental medicine.

INTRODUCTION

SYMPOSIUM PURPOSE

Since 1981, the International Symposium has been recognized as one of the most advanced medical forums in the world addressing the research and treatment of environmental effects on health and disease. The 2005 conference will focus on “The Autonomic Nervous System and Its Relationship to Environmental Pollutants Including the Cardiovascular System and Electromagnetic Sensitivity”. This Conference presents the most current information available while providing guidelines to identify, diagnose, treat and to prevent environmentally triggered responses in the body.

GOALS OF THE MEETING

- ! To provide new insights in *The Autonomic Nervous System and Its Relationship to Environmental Pollutants* and the environmental causes behind many problems you see.
- ! To present new diagnostic and treatment modalities to help you improve the quality of care for your complex patients.
- ! To provide concepts, tools that will enhance your practice.

OBJECTIVES OF THE MEETING

- ! Improve the outcome of treating patients with sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Use new concepts and treatments to help better diagnose and manage many patients with environmentally triggered problems and sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Apply the concepts of this conference to your practice by using nutrition and environmental manipulation for the treatment of sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Use the information presented to enhance the effectiveness, cost-efficiency, and competitiveness of the physician in relation to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.

INTENDED AUDIENCE

M.D.=s, D.O.=s, medical students, nurses, nutritionists and other health professionals interested in the concepts and practice of Environmental Medicine, Occupational Medicine and Toxicology.

EDUCATIONAL FORMATS

- ✂ Plenary
- ✂ Panels Discussions
- ✂ Case Studies

CONFERENCE FORMAT

The AEHF Committee has selected some of the leading experts in the fields of chronic disease, nutrition and chemical sensitivity.

Each speaker's presentation will last approximately 20 minutes and will be followed by a 10 minute question and answer session. All speakers are encouraged to use any and all appropriate audio/visual aids. (A brief outline of the speech is included in this booklet.)

GIVEN IN COOPERATION

William J. Rea, M.D., F.A.C.S.

Symposium Chairman,
American Environmental Health Foundation,
Environmental Health Center - Dallas,
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Bertie B. Griffiths, Ph.D.,

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Brody School of Medicine, East Carolina University
Department of Emergency Medicine
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**23rd Annual International Symposium
on Man and His Environment
Schedule**

Saturday, June 11, 2005

9:00 **ANNOUNCEMENTS/MODERATOR:** Bertie Griffiths, Ph.D.

9:05 “Modulation of Enteric Nervous System by Intestinal Contents”, Javier Santos, M.D.

9:25 Q & A

9:35 “Group A Strep and Neuro Psychiatric Disorders”, Richard Jaeckle, M.D.

9:55 Q & A

10:05 **MORNING BREAK WITH EXHIBITORS**

10:30 “Manifestations and Management of Mold Allergies”, David Hurst, M.D., Ph.D.

10:50 Q & A

11:00 “Treatment of the Damaged Autonomic Nervous System in Environmentally Damaged Patients”,
 William J. Rea, M.D., FACS

11:20 Q & A

11:30 “Intervention with Glutathione-Galavit; Realities of Delivery to Neurons”, Kaye Kilburn, M.D.

11:50 Q & A

12:00n **BUFFET LUNCH WITH EXHIBITORS**

MODERATOR: Richard Jaeckle, M.D.

1:30 “Recent Developments in Functional Imaging”, Theodore R. Simon, M.D.

1:50 Q & A

2:00 “Man's Sense of Awareness”, Jean Monro, M.D.

2:20 Q & A

2:30 “Controlled Inhalational Challenge in Multiple Chemical Sensitivity”, Roy Fox, M.D.

2:50 Q & A

3:00 **AFTERNOON BREAK WITH EXHIBITORS**

3:30 “The Sphenopalatine Ganglion and Environmental Sensitivity”, Dietrich Klinghardt, M.D., Ph.D.

3:50 Q & A

4:00 “Allergy as a Cause of Sleep Apnea Syndrome”, Jorge A. Ayala Moran, M.D.

4:20 Q & A

4:30 “Effect of EMF on Male Reproductive Organs”, Kou Sakabe, M.D.

4:50 Q& A

5:00 PANEL DISCUSSION/CASE STUDIES: Aristo Vojdani Ph.D.

“Neuroimmune Abnormalities in a Patient Exposed to a Combination of Mercury and Chemicals Used in Fumigation”

6:00 ADJOURN

SATURDAY, JUNE 11, 2005

ABSTRACTS

AND

HANDOUTS

Objectives & Notes

Javier Santos, M.D.

Date of talk: Saturday, June 11, 2005, 9:05am

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Board Certifications:

Not applicable

SPEECH TITLE: “**Modulation of Enteric Nervous System by Intestinal Contents**”

At the end of this Presentation, the participant should be able to:

1. To acknowledge that intestinal contents, particularly microbial products, may influence the activity and responses of enteric, autonomic and central nervous systems and innate and acquired immunity.
2. To preview how this influence could be relevant to the development of human inflammatory disorders.

The above information was provided by the Speaker.

Modulation of Enteric Nervous System by Intestinal Contents. Javier Santos, M.D., Ph.D. Hospital Universitario Valle de Hebron. Digestive System Research Unit. Barcelona, Spain.

Goals and Objectives

1. To acknowledge that intestinal contents, particularly microbial products, may influence the activity and responses of enteric, autonomic and central nervous systems and innate and acquired immunity.
2. To preview how this influence could be relevant to the development of human inflammatory disorders.

Outline

The intestinal epithelium is a single columnar layer with a surface area of about 400 m² where besides the enterocytes other cell types are present: goblet cells, endocrine cells, stem cells and intraepithelial immunocytes. Epithelial cells are bound together by junctional complexes in the apical surface that restrict the passage of even very small (2-kDa) molecules but and it is relatively impermeant to macromolecules or bacteria. The intestinal epithelium also boasts a number of specialized protective adaptations that are not found in other sites including anti-microbial peptides, secretory immunoglobulin A, mucins and Trefoil peptides. The apical surface of the enterocyt faces the intestinal lumen which homes a huge number of microorganisms, up to 100 trillion in distal segments, exceeding by far the number of all other microbial communities associated with the body's surfaces. The gut contains more than 100 million neurons with innervation extending through all layers of the tract, suggesting that this specific interface of nerves, microorganisms and immune cells might be crucial to normal homeostasis and to the prevention of uncontrolled inflammation

The gut-associated lymphoid tissue Peyer's patches is covered by a specialized follicle-associated epithelium containing M cells, a subepithelial dome rich in dendritic cells (DCs) and B-cell follicles that contain germinal centres. Transport of soluble proteins and microbes across the epithelium occurs through both specialized M cells and by DCs. Bacteria that penetrate the enterocyte epithelial layer are rapidly killed by the macrophages in the lamina propria. M cells use transepithelial vesicular transport to carry microbes to antigen presenting cells in the underlying gut-associated lymphoid tissue. The DC might extend its dendrite-like processes through epithelial tight junctions and sample and uptake luminal antigen directly. This process is uptake is regulated by CX3CL1, a chemokine produced by intestinal epithelial cells. TGF- β and other factors derived from stromal cells prompt resident intestinal macrophages to profound anergy. These macrophages do not express innate response receptors and do not release inflammatory cytokines in response to bacteria—but they retain phagocytic and bactericidal activity. Factors derived from epithelial cells condition intestinal dendritic cells to become nonresponsive although these cells are still able to open the tight junctions and sense of commensal bacteria. During infection, invasive bacteria activate epithelial cells to produce proinflammatory mediators that recruit additional immune cells.

A small number of bacteria can survive inside enabling the interaction of DCs with T and B cells in the Peyer's patches and/or the migration of DCs to the draining mesenteric lymph nodes. Although DCs loaded with commensal bacteria can traffic to the mesenteric lymph nodes, the lymph nodes function as a barrier, and the loaded DCs cannot penetrate farther to reach the systemic secondary-lymphoid tissues. The result is that the induction of immune responses by live bacteria is confined to the mucosa itself. Following activation, B- and T-cell blasts can leave the mesenteric lymph nodes through the efferent lymph, enter the bloodstream at the thoracic duct and home back to the intestinal mucosa.

The signalling loop that mediates the epithelial response to microorganisms is based on sensing of structural motifs known as pathogen-associated molecular patterns that are specific for prokaryotic components. The motifs are recognized by pattern-recognition receptors such as the Toll-like receptors or the nucleotide-binding oligomerization domain family of proteins. Although there are differences in the signalling pathways, activation of pattern-recognition receptors induces the expression of pro-inflammatory genes. This leads epithelial cells to produce an array of pro-inflammatory cytokines and chemokines, among which CXCL8 is most abundant.

The presence or absence of intestinal bacteria has a large impact on lymphoid structures of both the intestine and systemic tissues. The intestines of germ-free mice have low numbers of lamina propria CD4+ cells, greatly reduced numbers of IgA-producing cells and hypoplastic Peyer's patches. These abnormalities reverse within weeks of colonization. On the other hand, the hygiene hypothesis states that a leading cause of the increased incidence of allergy and inflammatory conditions in Western world is the decrease exposure to common infections during life. Two theories, immune deviation and counter regulation, offer explanations to simultaneous increase in autoimmunity and inflammatory disorders (Th1 mediated) and allergies (Th2 mediated) immune deviation and counter regulation although neither explanation is likely to account entirely for the long-term consequences of altered hygiene conditions

Finally, microbial endocrinology considers the possibility that eukaryotic and prokaryotic cells share common chemical messengers and receptors. If true, this may help to understand how a hormone could directly influence bacterial growth and also how the production of hormone-like compounds by microorganisms modulate host neuro-immune-cell responses.

Conclusion

Gut microflora actively communicates with the enteric and central nervous systems and the immune system to tightly regulate intestinal function

References

- 1.Science 2005;307:1915-1920.
- 2.Curr Op Immunol 2004, 16:277–283.
- 3.Nature Rev Immunol 2001;1:59-66.
- 4.Nature Rev Immunol 2004;4:481.

5. Nature Med 2005;11:254
6. Nat Rev Immunol 2004;4:962
7. Science 2002;296:4:
8. Gut 2005;54;317-320.
9. Trends Microbiol 2004;12:14
9. Trends Immunol 2004;25:505.
10. JPET 2005;312:417

**23rd Annual International Symposium
on Man and His Environment
Schedule**

Saturday, June 11, 2005

9:00 **ANNOUNCEMENTS/MODERATOR:** Bertie Griffiths, Ph.D.

9:05 “Modulation of Enteric Nervous System by Intestinal Contents”, Javier Santos, M.D.

9:25 Q & A

9:35 “Group A Strep and Neuro Psychiatric Disorders”, Richard Jaeckle, M.D.

9:55 Q & A

10:05 **MORNING BREAK WITH EXHIBITORS**

10:30 “Manifestations and Management of Mold Allergies”, David Hurst, M.D., Ph.D.

10:50 Q & A

11:00 “Treatment of the Damaged Autonomic Nervous System in Environmentally Damaged Patients”,
William J. Rea, M.D., FACS

11:20 Q & A

11:30 “Intervention with Glutathione-Galavit; Realities of Delivery to Neurons”, Kaye Kilburn, M.D.

11:50 Q & A

12:00n **BUFFET LUNCH WITH EXHIBITORS**

MODERATOR: Richard Jaeckle, M.D.

1:30 “Recent Developments in Functional Imaging”, Theodore R. Simon, M.D.

1:50 Q & A

2:00 “Man's Sense of Awareness”, Jean Monro, M.D.

2:20 Q & A

2:30 “Controlled Inhalational Challenge in Multiple Chemical Sensitivity”, Roy Fox, M.D.
2:50 Q & A

3:00 AFTERNOON BREAK WITH EXHIBITORS

3:30 “The Sphenopalatine Ganglion and Environmental Sensitivity”, Dietrich Klinghardt, M.D., Ph.D.
3:50 Q & A

4:00 “Allergy as a Cause of Sleep Apnea Syndrome”, Jorge A. Ayala Moran, M.D.
4:20 Q & A

4:30 “Effect of EMF on Male Reproductive Organs”, Kou Sakabe, M.D.
4:50 Q& A

5:00 PANEL DISCUSSION/CASE STUDIES: Aristo Vojdani Ph.D.
“Neuroimmune Abnormalities in a Patient Exposed to a Combination of Mercury and Chemicals Used in Fumigation”

6:00 ADJOURN

SATURDAY, JUNE 11, 2005

ABSTRACTS

AND

HANDOUTS

Objectives & Notes

Javier Santos, M.D.

Date of talk: Saturday, June 11, 2005, 9:05am

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Residency:

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Board Certifications:

Not applicable

SPEECH TITLE: **“Modulation of Enteric Nervous System by Intestinal Contents”**

At the end of this Presentation, the participant should be able to:

3. To acknowledge that intestinal contents, particularly microbial products, may influence the activity and responses of enteric, autonomic and central nervous systems and innate and acquired immunity.
4. To preview how this influence could be relevant to the development of human inflammatory disorders.

The above information was provided by the Speaker.

Modulation of Enteric Nervous System by Intestinal Contents. Javier Santos, M.D., Ph.D. Hospital Universitario Valle de Hebron. Digestive System Research Unit. Barcelona, Spain.

Goals and Objectives

1. To acknowledge that intestinal contents, particularly microbial products, may influence the activity and responses of enteric, autonomic and central nervous systems and innate and acquired immunity.
2. To preview how this influence could be relevant to the development of human inflammatory disorders.

Outline

The intestinal epithelium is a single columnar layer with a surface area of about 400 m² where besides the enterocytes other cell types are present: goblet cells, endocrine cells, stem cells and intraepithelial immunocytes. Epithelial cells are bound together by junctional complexes in the apical surface that restrict the passage of even very small (2-kDa) molecules but and it is relatively impermeant to macromolecules or bacteria. The intestinal epithelium also boasts a number of specialized protective adaptations that are not found in other sites including anti-microbial peptides, secretory immunoglobulin A, mucins and Trefoil peptides. The apical surface of the enterocyt faces the intestinal lumen which homes a huge number of microorganisms, up to 100 trillion in distal segments, exceeding by far the number of all other microbial communities associated with the body's surfaces. The gut contains more than 100 million neurons with innervation extending through all layers of the tract, suggesting that this specific interface of nerves, microorganisms and immune cells might be crucial to normal homeostasis and to the prevention of uncontrolled inflammation

The gut-associated lymphoid tissue Peyer's patches is covered by a specialized follicle-associated epithelium containing M cells, a subepithelial dome rich in dendritic cells (DCs) and B-cell follicles that contain germinal centres. Transport of soluble proteins and microbes across the epithelium occurs through both specialized M cells and by DCs. Bacteria that penetrate the enterocyte epithelial layer are rapidly killed by the macrophages in the lamina propria. M cells use transepithelial vesicular transport to carry microbes to antigen presenting cells in the underlying gut-associated lymphoid tissue. The DC might extend its dendrite-like processes through epithelial tight junctions and sample and uptake luminal antigen directly. This process is uptake is regulated by CX3CL1, a chemokine produced by intestinal epithelial cells. TGF- β and other factors derived from stromal cells prompt resident intestinal macrophages to profound anergy. These macrophages do not express innate response receptors and do not release inflammatory cytokines in response to bacteria—but they retain phagocytic and bactericidal activity. Factors derived from epithelial cells condition intestinal dendritic cells to become nonresponsive although these cells are still able to open the tight junctions and sense of commensal bacteria. During infection, invasive bacteria activate epithelial cells to produce proinflammatory mediators that recruit additional immune cells.

A small number of bacteria can survive inside enabling the interaction of DCs with T and B cells in the Peyer's patches and/or the migration of DCs to the draining mesenteric lymph nodes. Although DCs loaded with commensal bacteria can traffic to the mesenteric lymph nodes, the lymph nodes function as a barrier, and the loaded DCs cannot penetrate farther to reach the systemic secondary-lymphoid tissues. The result is that the induction of immune responses by live bacteria is confined to the mucosa itself. Following activation, B- and T-cell blasts can leave the mesenteric lymph nodes through the efferent lymph, enter the bloodstream at the thoracic duct and home back to the intestinal mucosa.

The signalling loop that mediates the epithelial response to microorganisms is based on sensing of structural motifs known as pathogen-associated molecular patterns that are specific for prokaryotic components. The motifs are recognized by pattern-recognition receptors such as the Toll-like receptors or the nucleotide-binding oligomerization domain family of proteins. Although there are differences in the signalling pathways, activation of pattern-recognition receptors induces the expression of pro-inflammatory genes. This leads epithelial cells to produce an array of pro-inflammatory cytokines and chemokines, among which CXCL8 is most abundant.

The presence or absence of intestinal bacteria has a large impact on lymphoid structures of both the intestine and systemic tissues. The intestines of germ-free mice have low numbers of lamina propria CD4⁺ cells, greatly reduced numbers of IgA-producing cells and hypoplastic Peyer's patches. These abnormalities reverse within weeks of colonization. On the other hand, the hygiene hypothesis states that a leading cause of the increased incidence of allergy and inflammatory conditions in Western world is the decrease exposure to common infections during life. Two theories, immune deviation and counter regulation, offer explanations to simultaneous increase in autoimmunity and inflammatory disorders (Th1 mediated) and allergies (Th2 mediated) immune deviation and counter regulation although neither explanation is likely to account entirely for the long-term consequences of altered hygiene conditions

Finally, microbial endocrinology considers the possibility that eukaryotic and prokaryotic cells share common chemical messengers and receptors. If true, this may help to understand how a hormone could directly influence bacterial growth and also how the production of hormone-like compounds by microorganisms modulate host neuro-immune-cell responses.

Conclusion

Gut microflora actively communicates with the enteric and central nervous systems and the immune system to tightly regulate intestinal function

References

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- 2.Curr Op Immunol 2004, 16:277–283.
- 3.Nature Rev Immunol 2001;1:59-66.
- 4.Nature Rev Immunol 2004;4:481.

5. Nature Med 2005;11:254
6. Nat Rev Immunol 2004;4:962
7. Science 2002;296:4:
8. Gut 2005;54;317-320.
9. Trends Microbiol 2004;12:14
9. Trends Immunol 2004;25:505.
10. JPET 2005;312:417

Objectives & Notes

Richard Jaeckle, M.D.

Date of talk: Saturday, June 11, 2005, 9:35am

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Training:

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Medical School/ University Attended

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Internship:

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Residency:

Psychiatry: St Louis Univ Hospitals & Child
Psychiatry: Washington Univ Child Guidance
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Board Certifications:

AmerBdPsyNeurol:Psychiatry; AmerBdPsyNeurol:
Child Psychiatry; AmerBdEnvironMed

SPEECH TITLE: "Group A Strep and Neuro Psychiatric Disorders"

At the end of this Presentation, the participant should be able to:

1. Know the definition and pathogenicity of PANDAS.
2. Anticipate the incidence of GAS in mood/behavior disorders
3. Associate certain psychiatric disorders to medical problems

The above information was provided by the Speaker.

Richard G Jaeckle, MD

8220 Walnut Hill #404

Dallas, TX 75231

Group A Strep and NeuroPsychiatric Disorders

Objectives of Presentation:

- 1) Know the Definition and Pathogenicity of PANDAS.
- 2) Anticipate the Incidence of Group A Strep (GAS) in NeuroPsychiatric Disorders.
- 3) Associate Certain Psychiatric disorders to Medical Problems.

Outline:

The initial description and definition of PANDAS in 1994 is followed by the case presentation of a teenager with depression and schizophrenia whose illness was associated with GAS. An interest in the association of GAS and mood disorders led to the study of 100 healthy psychiatric patients for indications of persistent colonization with GAS and elevations of the anti-streptolysin O antibody (ASO). The PANDAS profile was used in some patients. When possible, tonsillar size, rapid ID throat swabs and skin test with the Group A Strep antigen was also performed. The incidence of positive index patients is 29% and 10% of the cohort received tonsillectomy and adenoidectomy.

Conclusions:

- There appears to be a high incidence of GAS colonization in these psychiatric patients.
- Rapid Strep ID is not an effective tool for detecting GAS colonization.
- Tonsillar size is not an reliable tool for detecting colonization
- ASO titer is a simple and effective tool for detecting GAS colonization.
- Skin test with GAS vaccine is not commercially available, but is quite sensitive and useful.
- Significant medical problems are not being recognized and treated.
- The PANDAS panel provides useful additional parameters for evaluation of GAS pathogenicity

References:

- Bowers M, Will Immunotherapy succeed whether others have failed Neuropsychiatry reviews, v2#2, Mar 2001
- <http://www.nimh.nih.gov/research.pandassummary.cfm>
– Aug18, 2001; NIMH Roundtable
- Arch Pediatr Adol Med 2002;156(4)356-361
- <http://intramural.nimh.nih.gov/research/pdn/web.htm>
- Leonard HL, Swede SE, PANDAS; Int J Neuropsychopharm 4:191, 2001
- Vojdani A et al, Antibodies to Neuron-specific Antigens in children with autism: Possible cross-reaction with encephalitogenic proteins from milk, chlamydia pneumoniae and GAS; Neuroimmun 129:168, 2002

Objectives & Notes

David Hurst, M.D., Ph.D.

Date of talk: Saturday, June 11, 2005, 10:30am

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Training:

Current Job Description:	Private Practice – Otolaryngic Allergy
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Medical School/University Attended:	Indiana University
Internship:	Portland, ME
Residency:	Tufts – B.U. – Boston, Massachusetts
Board Certifications:	AAO-UNS, AAOA
Other Information:	Ph.D. – Uppgala, Sweden

SPEECH TITLE: “Manifestations and Management of Mold Allergies”

At the end of this Presentation, the participant should be able to:

1. Recognize the unusual symptoms of confusion and fatigue and chronic external otitis which may be signs of mold allergy.
2. Understand how mold allergy affects sinusitis and asthma.
3. Be comfortable in knowing how to manage patients with multiple mold inhalant and food allergies.

The above information was provided by the Speaker.

Manifestation and Management of Mold Allergies

David S. Hurst, M.D., Ph.D.
Portland, ME

Abstract:

This course will briefly survey the emerging concepts regarding parasympathetic manifestations in allergy. Discussion will center on the diverse spectrum of symptoms encountered in patients with mold allergy including classic rhinitis, chronic otitis externa, asthma, eczema and complaints of bizarre mood and behavior disorders among both children and adults. The efficacy of mold immunotherapy and mold food elimination diets will be reviewed.

References:

(PMID = Pub Med ID)

General Allergy

Hurst DS, Gordon BR, Fornadley JA, Hunsaker DH.
Safety of home-based and office allergy immunotherapy: A multicenter prospective study.
Otolaryngol Head Neck Surg. 1999 Nov;121(5):553-61.
PMID: 10547469

Passalacqua G, Canonica GW. *****
Long-lasting clinical efficacy of allergen specific immunotherapy.
Allergy. 2002 Apr;57(4):275-6.
PMID: 11906355

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Treating the allergic patient: think globally, treat globally.
Allergy. 2002 Oct;57(10):876-83. "Unified Airway Disease"
PMID: 12269932

Smith GC, Pell JP. (A *great spoof on evidenced based medicine*) *****
Parachute use to prevent death and major trauma related to gravitational challenge: **Systematic review of randomised controlled trials.**
BMJ. 2003 Dec 20;327(7429):1459-61. Review.
PMID: 14684649

Nomenclature

Johansson SG, Bieber T, Dahl R, Friedmann PS, Lanier BQ, Lockey RF, Motala C, Ortega Martell JA, Platts-Mills TA, Ring J, Thien F, Van Cauwenberge P, Williams HC.

Revised nomenclature for allergy for global use: Report of the Nomenclature Review Committee of the World Allergy Organization, October 2003.

J Allergy Clin Immunol. 2004 May;113(5):832-6. PMID: 15131563

A revised nomenclature for allergy. An EAACI position statement from the EAACI nomenclature task force.

Allergy. 2001 Sep;56(9):813-24. Review.

PMID: 11551246

David Hurst, MD, PhD
207-883-6464

General Sinusitis/Asthma

Mold Allergy

Hossain MA, Ahmed MS, Ghannoum MA.

Attributes of *Stachybotrys chartarum* and its association with human disease.

J Allergy Clin Immunol. 2004 Feb;113(2):200-8; quiz 209.

Review. PMID: 14767429

Fungal Allergy and Pathogenicity; *ed: Adorini, Arai, Berek, Schmitt-Verhulst*

In: Chem Immunol. 2002;81:5-9. Review.

PMID: 12102005

Mold allergy: some progress made, more needed.

J Allergy Clin Immunol. 2004 Feb;113(2):216-8. No

abstract available. PMID: 14767432

Are indoor molds causing a new disease?

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Jan 09. Review.

PMID: 14722497

Campbell AW, Thrasher JD, Madison RA, Vojdani A, Gray MR, Johnson A.

Neural autoantibodies and neurophysiologic abnormalities in patients exposed to molds in water-damaged buildings.

Arch Environ Health. 2003 Aug;58(8):464-74.

PMID: 15259425

Dennis DP.

Chronic sinusitis: defective T-cells responding to superantigens, treated by reduction of fungi in the nose and air.

Arch Environ Health. 2003 Jul;58(7):433-41.

PMID: 15143856

Johanning E.

Indoor moisture and mold-related health problems.

Allerg Immunol (Paris). 2004 May;36(5):182-5. Review.

PMID: 15206571

Bush RK, Portnoy JM.

The role and abatement of fungal allergens in allergic diseases.

J Allergy Clin Immunol. 2001 Mar;107(3 Suppl):S430-40.

Review. PMID: 11242604

more info and handouts:

<http://home.earthlink.net/~mear/dr-davi-dhurst/>

Objectives & Notes

William J. Rea, M.D., FACS

Date of talk: Saturday, June 11, 2005, 11:00am

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Current Faculty Appointments:	Professor of Medicine, Capital University of Integrative Medicine, Washington, D.C.
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Internship:	Parkland Memorial Hospital, Dallas, TX
Residency:	University of Texas SW Medical School, Dallas, TX
Board Certifications:	American Board of Surgery; American Board of Thoracic Surgery; American Board of Environmental Medicine
Other Information:	“Optimum Environments for Optimum Health and Creativity”

SPEECH TITLE: “Autonomic Nervous System Changes with Treatment in 100 Chemically Sensitvite Patients”

At the end of this Presentation, the participant should be able to:

1. Treat the nutritional aspects of ANS dysfunction
2. Treat the toxic aspects of ANS dysfunction
3. Treat the hypersensitivity aspects of ANS dysfunction

The above information was provided by the Speaker.

Objectives & Notes

Kaye H. Kilburn, M.D.

Date of talk: Saturday, June 11, 2005, 11:30am

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Residency:	University of Utah Medicine, Pathology; Duke, Cardiopulmonary Physiology; University of London Cardiology
Board Certifications:	Diplomat American Board of Internal Medicine, American Board of Preventive Medicine Occupational Health
Other Information:	Books: <i>Chemical Brain Injury</i> and <i>Endangered Brains</i> <i>Mold and Mycotoxins</i> , (Kilburn, KH ed) Written several articles

SPEECH TITLE: “Intervention with Glutathione-Galavit; Realities of Delivery to Neurons”

At the end of this Presentation, the participant should be able to:

1. Understand the rational basis for redox regulation of brain cells.
2. See the need for measurement of brain functions before and intervention.
3. Have basis for evaluating therapy directed to brain impairment and chemical intolerance.

The above information was provided by the Speaker.

**Intervention in CBI-MCS with Glutathione–Galavit
Realities in Delivery to Neurons**

¹**Kaye H. Kilburn, M.D.**

²**William S. Lynn, Ph.D.**

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Background

Empirical treatment for chemical brain injury especially that due to molds and mycotoxins and multiple chemical sensitivity beyond “to avoid” includes body burden reduction by purging, sweating, exercise and desensitization. Many nutritional supplements and anti oxidants, statins, antifungal agents and immune enhancers have been given to patients. Often patients “feel better” during or after these ministrations but there are no measurements of neurobehavioral function to appraise efficacy. Furthermore glutathione (50 to 100 mg/ml) to provide sulfhydryl SH groups has a logical promise and has been administered without adverse effects to many patients with multiple chemical sensitivity.

The initial objective was to determine the effect of intranasal glutathione (G_1) on neurobehavioral (NB) function in patients with chemical intolerance and chemical brain injury. Several physicians had described improvement in patients’ feelings and decreased symptoms from 1 or 2 squirts of glutathione, 100 mg/ml in each nostril 3 times a day. The procedure adopted after finding only one of 30 patients reacted adversely was to obtain baseline measurements of 26 NB functions, teach patients to self administer G_1 and repeat measurements after intervals of one month after that (TW, JL, MC, JM). Four patients returned at monthly or bimonthly intervals for 4 to 12 months. Some functions improved, but rarely to predicted (normal) levels but subjective recall, memory, mood, and alertness improved and symptom frequencies decreased. There was no toxicity, but no additional improvements after 30 days, in fact none after 14 days so a shorter interval of 3 days was adopted for evaluation of G_1 . The second step was to add an oxidation-reduction regulating, redox, agent Galavit (G_a) 10 mg/ml and continue G_1 at 50 mg/ml for 3 days (G_1 / G_a). All 4 patients decreased their neurobehavioral abnormalities and feelings and symptoms improved after 3 days. The third step was to measure quickness of response and then fourth to see whether G_a alone improved functions.

Objective

This study was designed .to test whether functions changed during or after IN glutathione and compare effects with an IN redox agent α -luminol (Galavit).

Methods

Patients were evaluated for 26 neurobehavioral functions including balance, reaction time, color determination, visual field performance and score, hearing, vibration and grip strength.

Cognitive tests included problem solving in Culture Fair, digit symbol vocabulary, recall of stories (memory), peg placement, trailmaking A and B and information similarities and picture completion, Profile of Mood States, assessed feeling supplemental by Beck’s Depression Scale and the McLean Limbic System Inventory. A well seasoned machine readable constrained data base facilitated data handling and comparisons.

Glutathione was initially given by aerosol but as equivalent effects on symptoms followed the intranasal administration, 27 patients received approximately 50 or 100 mg per day intranasal via sniffing that delivered 1.0 to 1.20 ml per day to the nose. The efficiency of nasal absorption to the brain is unknown but insulin, oxytocin and peptides have been measured in cerebrospinal fluid after sniffing them. Baseline measurements of 26 functions provide the baseline for effects of aerosol – intranasal glutathione. Patients returned for testing after 4 or 6 weeks.

Results: In a year's interval since first evaluation, two patients of 27 had returned to normal function. Interval function of the other 25 was unchanged or worse. IN glutathione improved function, reduced abnormality score by 2-3 with greater effect on psychological than physiological performance with no effect on balance and reaction time. One subject who had been exposed to organophosphate insecticides became nauseated and vomited after the first dose of IN glutathione at 50 mg/ml and could not tolerate a reduced dose.

The second step was to add a redox regulator to IN glutathione. We chose α -luminol, manufactured to drug purity in Russia as Galavit that has been used to treat inflammation and cancer by injections into tissue and intravenously. No toxic effects were seen when it was taken by the investigators and given to two patients. Ten patients were in this search for efficacy testing. They mixed Galavit 20 mg/ml with Glutathione 100 mg/ml and 6 subjects administered it intra nasally 3 times per day. Four patients showed decreased neurobehavioral abnormalities after 3 days with improved feeling state (POMS score) and decreased symptoms.

The third step, four new patients, 2 untreated and 2 who received G_I without improvement earlier were given G_a alone and were measured at 24 and 48 hours. Three improved at 24 hours and improved further at 48 hours. Total abnormalities decreased in DM from base of 8 to 5 and 5; JK from base of 14 to 10 and 3.5 and TT from base of 6.5 to 5.5 to 6 but balance and reaction time improved. Further improvement was seen in 2 after glutathione (G_I) was added for 24 and 48 hours, DM after G_I/ G_a was 1 and remained 1 and TT from base of 6.5, to 5.5 and 6.0, and after G_I/ G_a was 6.0. DM had normal function and no mood or symptom problems and remained normal after 2 weeks at home. TT had improved balance that remained abnormal. The fourth, SM, was unchanged so returned for a second course. The third, JK who could not take glutathione, was almost functionally normal after 48 hours of Galavit. Abnormalities decreased for baseline of 14 to 3.5.

Summary:

a. Neurobehavioral improvement after Galavit (G_a) occurred in 24 to 48 hours in 4/4 (DM, TW, JK, TT)

b. Intranasal glutathione added to Galavit (G_I/G_a) increased effect in 6 of 6 (JL, MC, DM, TW, JB, FB). Two patients, FB and JB, had least improvement. JB, age 19 years had had brain surgery twice to remove seizure foci that were attributed to mold/mycotoxin impairment.

c. Two of 3 patients with balance disorders improved markedly (TW, and JM) while TT, the most abnormal, had some improvement in extremely abnormal balance but greatly improved cognitive mood and feeling state.

d. After stopping Galavit, DM remained rehabilitated, TW crashed, but was restored with G_I/ G_a . and JM improved again with G_I/ G_a .

e. One patient, SM, had no improvement on G_1 alone, G_a alone, or for 48 hours following the combination of G_1/ G_a . but after Glutathione for 2 weeks took Galavit for 4 days with improvement almost to normal. This defined him as a slow responder.

Conclusion:

1. Galavit (α -luminol) given intranasally improved functions including balance.
2. Glutathione is synergistic with Galavit
3. Balance is a key function for evaluating therapy.
4. Redox regulators combined with SH or NO agents deserve further investigation.

Objectives & Notes

Richard Jaeckle, M.D.

Date of talk: Saturday, June 11, 2005, 1:00pm

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Training:

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Medical School/ University Attended	University of Texas Southwestern Medical School
Internship:	Veterans Administration Hospital, Dallas, TX
Residency:	Psychiatry: St Louis Univ Hospitals & Child Psychiatry: Washington Univ Child Guidance Clinic
Board Certifications:	AmerBdPsyNeurol:Psychiatry; AmerBdPsyNeurol: Child Psychiatry; AmerBdEnvironMed

SPEECH TITLE: “Group A Strep and Neuro Psychiatric Disorders”

At the end of this Presentation, the participant should be able to:

4. Know the definition and pathogenicity of PANDAS.
5. Anticipate the incidence of GAS in mood/behavior disorders
6. Associate certain psychiatric disorders to medical problems

The above information was provided by the Speaker.

Richard G Jaeckle, MD

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Group A Strep and NeuroPsychiatric Disorders

Objectives of Presentation:

- 4) Know the Definition and Pathogenicity of PANDAS.
- 5) Anticipate the Incidence of Group A Strep (GAS) in NeuroPsychiatric Disorders.
- 6) Associate Certain Psychiatric disorders to Medical Problems.

Outline:

The initial description and definition of PANDAS in 1994 is followed by the case presentation of a teenager with depression and schizophrenia whose illness was associated with GAS. An interest in the association of GAS and mood disorders led to the study of 100 healthy psychiatric patients for indications of persistent colonization with GAS and elevations of the anti-streptolysin O antibody (ASO). The PANDAS profile was used in some patients. When possible, tonsillar size, rapid ID throat swabs and skin test with the Group A Strep antigen was also performed. The incidence of positive index patients is 29% and 10% of the cohort received tonsillectomy and adenoidectomy.

Conclusions:

- There appears to be a high incidence of GAS colonization in these psychiatric patients.
- Rapid Strep ID is not an effective tool for detecting GAS colonization.
- Tonsillar size is not an reliable tool for detecting colonization
- ASO titer is a simple and effective tool for detecting GAS colonization.
- Skin test with GAS vaccine is not commercially available, but is quite sensitive and useful.
- Significant medical problems are not being recognized and treated.
- The PANDAS panel provides useful additional parameters for evaluation of GAS pathogenicity

References:

- Bowers M, Will Immunotherapy succeed whether others have failed Neuropsychiatry reviews, v2#2, Mar 2001
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– Aug18, 2001; NIMH Roundtable
- Arch Pediatr Adol Med 2002;156(4)356-361
- <http://intramural.nimh.nih.gov/research/pdn/web.htm>
- Leonard HL, Swede SE, PANDAS; Int J Neuropsychopharm 4:191, 2001
- Vojdani A et al, Antibodies to Neuron-specific Antigens in children with autism: Possible cross-reaction with encephalitogenic proteins from milk, chlamydia pneumoniae and GAS; Neuroimmun 129:168, 2002

Objectives & Notes

Theodore R. Simon, M.D.

Date of talk: Saturday, June 11, 2005, 1:30pm

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Training:

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Residency:	University of California at San Francisco; Yale
Board Certifications:	American Board of nuclear Medicine

SPEECH TITLE: “Recent Developments in Functional Imaging”

At the end of this Presentation, the participant should be able to:

1. Identify new diagnostic options with functional imaging.
2. Understand the requirements imposed on patients for functional imaging.
3. Understand ordering issues for obtaining functional imaging.

The above information was provided by the Speaker.

Recent Developments in Functional Imaging
by Theodore R. Simon, M.D.

Goals and Objectives

The participant should understand the strategic implications of functional imaging. These strategies will be explored to allow the participant to become familiar with how to use them to effectively develop diagnostic assessments and objective measures of therapeutic efficacy.

Outline

We will examine available functional imaging techniques to identify and quantitate various attributes of cell function. Specifically we will address central and autonomic neurotransmitters, effects on cell behavior, kinetics, receptors, and metabolism. Case studies of these modalities will be provided, to describe the clinical milieu in which the practitioner can obtain valuable information.

Attention will be directed toward the types of examinations that are available. The tracers, imaging devices, and data analyses will be covered in detail. Tracers currently available will be emphasized, but a peek at likely imminent developments will also be provided. Imaging devices will be explained, especially in light of current widespread confusion regarding PET/CT and SPECT/CT. Examples of one-, two-, three-, and four-dimensional image analyses will be explained as they relate to gathering functional information.

Special consideration will be given to explaining the likely experience of the patient in order to provide the practitioner with a sense of the implications of orders for these tests.

Conclusions

Efficient use of functional imaging can provide both highly sensitive and highly specific information that can target a diagnosis. Moreover, having made the diagnosis and embarked on the therapeutic regimen, functional imaging can objectively assess the success of the strategy and provide prognostic data to provide the patient with reasonable expectations of the likelihood of improvement.

Objectives & Notes

Jean Monro, M.D.

Date of talk: Saturday, June 11, 2005, 2:00pm

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Residency:	London Hospital
Board Certifications:	MB, BS, MRCS, LRCP, FAAEM, DipIBEM, MACOEM
Other Information:	Treatment of cancer with mushroom products. Arch environ Health 2003; 58:533-7

SPEECH TITLE: “Man's Sense of Awareness”

At the end of this Presentation, the participant should be able to:

1. Appreciate that particles and frequencies are perceived by man;
2. Realize that the pathway for these is neural;
3. Understand the interaction between sensitivities and allergies and why neutralization therapy works for both.

The above information was provided by the Speaker.

Breakspear Hospital

MAN'S SENSE OF AWARENESS

In any individual with a disease, there are 3 main categories which are amenable to treatment:

- infectious agents
- nutritional state
- pollutants

These have to be considered for each individual in the light of their own immunology, their own encounters and their own nutritional intake.

With regard to our state of health, this is primarily dependent on an individual's discrimination between self and non-self, which is learned *in utero*. Thereafter, what is non-self, and which we need to be incorporated into the body, we tag with a piece of self (Secretory IgA). This is then accepted in the body without reaction.

Secretory IgA is a non-inflammatory antibody and reduces the body's other reactions to any foods that are admitted with it. If there is inadequate Secretory IgA and a food gains access to the system, then the body will react with reactive antibodies to disperse a possible threat. If, however, the substance is not required by the body, but still has IgA attached to it, it can be broken down in the liver and many bacteria and bacterial products are dispersed without untoward effect.

The pathways for foreign material, regarded as “suspicious” by the immune system and not required for sustenance, are two-fold:

- 1 Cell-mediated immunity
- 2 Humoral immunity

These two pathways are antagonistic towards each other, as resources have to be carefully directed and husbanded when there is a threat to the body. If, for example, there is a massive bacterial infection or viral infection, this can be targeted by cell-mediated immunity and antibody production. If, however, there is a very large parasite, such as a worm, infecting the body, then clearly cells are not going to be able to encompass this and antibodies are made, which is the fluid (humoral) immunity. The protein messengers, which perpetuate this type of reaction, are called cytokines. They work in groups to direct the reactions; cytokine groups are known as Th1 and Th2. The group producing cell-mediated immunity are called Th1 cytokines. The group producing humoral immunity are called Th2 cytokines; these induce humoral/fluid/antibody reaction. We know that in people who have long-term viral infections there is a cytokine shift from Th1 to Th2. When this happens, we have to be aware that excessive antibody production results in allergies.

There is a distinction between allergy and sensitivity. The pathway for allergy is subject to many reactions with cells. The pathway for sensitivity is also involved in allergic states, but is the neural pathway for both allergy and sensitivity.

The universal means of perceiving identity is to recognise things which are not self. We do this through our perception of weak electromagnetic fields. If we were to be bombarded by magnetic fields which were intense, this would be the equivalent of being overwhelmed by information. It is not how perception works. We have the means of identifying very weak electromagnetic fields and assessing these as part of our non-self environment. Every item with which we are in touch has a weak electromagnetic field. To be able to sense this and discern what is appropriate for us and what is dangerous is a universal phenomenon of mankind, as basic as the sense of smell or hearing in higher animals. Where there is disequilibrium in our means of assessment of this, people require help. This is the point at which foods, chemicals, inhalants and electromagnetic frequencies, beyond those which can be tolerated, disturb the individual and this is why we use neutralising vaccines for our patients. The amplification of this follows.

Just as light frequencies are converted into chemicals in the eye, then transmitted through ionic exchange via the optic nerve to the ophthalmic area of the cortex, so our electromagnetic perception is similarly mediated.

The other special senses have receptors, physical information which becomes transformed through chemical intervention then through swift ionic exchanges to recognition in the cortex. Sound, touch, vibration, position, temperature are all perceived thus. Another aspect of physics and chemistry co-operating in perception is that melatonin is formed in the pineal gland in the dark. Its production can be switched off by light whether in the sighted or the blind. The blind cannot perceive through sightless eyes or through the reception of frequencies through the eyes. However, it is very likely that they can react to frequencies perceived through the skin.

It is known that the supra-optic nuclei are involved in this perception and jetlag can be overcome by their response to light through a non-optic nerve channel. Our sense of smell is said to be recognition of molecules at the molecular level. However, pheromones are perceived by animals without such a major organisation as the nose; for example, creatures with antennae can appreciate these.

We receive information through perception of the whole range of electromagnetic frequencies through the skin and mucous membranes by the dendritic cells, thence via the C-fibres to the autonomic nervous system, the cord, the hypothalamus and cortex.

We have proved that people who have sensitivities are able to perceive foods, chemicals, inhalants and electromagnetic frequencies similarly. All the first three, if diluted, exhibit the phenomenon of hormesis. This is the stimulatory effect of small doses of substances which, in larger doses, are inhibitory.

With our neutralising vaccines, hormesis is exhibited. We have demonstrated that symptoms produced by a series of doses of a sequence of decreasing dilutions can be negated at one point in the series. Another bipolar response curve will then occur with further dilutions. These continue through a range of dilutions beyond Avogadro's number. The weaker the range of dilutions, the greater the induction of symptoms and the greater the interval between the provocation and the nullification of symptoms.

This series of provocation and neutralisation strengths can be applied to the individual either by:

- | | |
|------------------------------|--|
| 1) subcutaneous injection | 5) application with a phial against the skin |
| 2) intradermal injection | 6) application by a phial held away from |
| 3) sublingual administration | the skin at varying distances |
| 4) cutaneous application | 7) a weak electromagnetic frequency |

The explanation of all of these means of inducing and nullifying symptoms is that the receptor is receiving not a chemical, but a physical initiation of response. Patients exposed to frequencies through the entire frequency range from 1 Hz to 2 GHz in a Faraday cage have shown exactly the same provocation and neutralisation of symptoms.

The receptor is the dendritic cell. It is connected to a C-fibre, then to the autonomic nervous system, the spinal cord, hypothalamus and cortex, where the appreciation of the frequency is that of awareness of self and awareness of non-self, in that the frequencies of all objects outside the body are distinct. We see symptoms induced in patients at any of these intersections.

We see:

- 1 Cutaneous reactions equivalent to a histamine response.
- 2 C-fibre transmission, which can be demonstrated. It can be blocked by transcutaneous field applications.
- 3 Effects on the autonomic nervous system - we know that anaphylaxis can occur with autonomic system inhibition of response (how else can a peanut in someone's pocket at a doorway to a room be perceived by a highly sensitive patient within that room, as we have observed?). The standard view is an antigen antibody response, which could be mitigated through reception of frequencies. We can demonstrate any autonomic nervous system effect, either sympathetic or parasympathetic, with dilutions of vaccines, including changes in pulse rate, asthmatic effects or

rhinitis which can be switched on and off and abdominal distension, due to parasympathetic effects.

- 4 Effects on the spinal cord - we have observed instant weakness of a limb, with difficulty in walking, or weakness of an arm, for example.
- 5 Changes in emotional states - because hypothalamic effects can be switched on and off very swiftly.
- 6 Reception of the information of the whole electromagnetic perception system by the cortex allows awareness of self and non-self.

All of these have been demonstrated in varying degrees in 12,000 patients. We have captured many of these effects on video film. The effects can be replicated and demonstrated independently to any observer and are therefore valid. For any individual, the symptoms produced may be identical with different antigenic stimuli. We can observe these responses in any individual with a degree of sensitivity, because the responses are the universal property of mankind. Sensitivity is heightened in the ill. This is, therefore, our electromagnetic perception system and is responsible for awareness of foreign material.

Jean A Monro

Medical Director

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Objectives & Notes

Roy Fox, M.D.

Date of talk: Saturday, June 11, 2005,
2:30pm

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Training:

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Current Faculty Appointments:	Professor of Medicine; Dalhousie University; N.S., Canada
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Internship:	Newcastle Teaching Hospitals
Residency:	Royal Free Hospital, London, England
Board Certifications:	FRCP (U.K. – Int. Medicine); FRCPC (Gastroenterology)
Other Information:	Master of Environmental Studies (Dalhousie) 2001 – Thesis “Multiple Chemical Sensitivity and the Environment”: “Env. Sensitivities in Dialysis Unit” Indoor Air 1999. ‘Environment & MCS’ Indoor Air 2002: “Adaptation in Individuals with Heightened Sensitivity to Combined Environmental Factors” Archives of Complex Env. Studies 2003.

SPEECH TITLE: “Controlled Inhalational Challenge in Multiple Chemical Sensitivity”

At the end of this Presentation, the participant should be able to:

1. Experimental set up to identify reaction to chemicals in MCS.
2. Problems with adaptation to changes in environment in patients with MCS
3. Complexity of reactivity to environmental triggers

The above information was provided by the Speaker.

CONTROLLED INHALATIONAL CHALLENGE IN MULTIPLE CHEMICAL SENSITIVITY (MCS)

Roy Fox ¹, Michel Joffres ^{1,2}, Tara Sampalli ¹

¹ Nova Scotia Environmental Health Centre, Fall River, Nova Scotia, Canada

² Community Health and Epidemiology, Faculty of Medicine, Dalhousie University, Halifax, Canada

Reactivity to environmental triggers in patients with MCS is complex, and it is clear that the reactivity is not usually allergic in nature or due to classical toxic damage. Much discussion has centered on the role of other factors, such as fear, societal fear of chemicals and anxiety.

Patients with MCS have been challenged with chemicals in various studies, but most such studies have not accounted for the autonomic dysfunction and compounding factors such as anxiety and fear. A challenge booth was constructed at the Nova Scotia Environmental Health Centre, a very clean and specially constructed building. The design allows common chemicals to be introduced into the air supply, and for the patient or observing nurse to be unaware of the introduction of a chemical or placebo. In a pilot study of 12 patients with MCS and 7 control subjects it was found that all the control subjects readily adapted to the baseline experimental protocol within 1 or 2 sessions, with 86% adapting in 1 session. After 4 sessions, 2 of the patients could not adapt and still showed random changes in skin conductance and electromyography measured at the upper trapezius. Of the remaining 10 subjects, the number of sessions required for adaptation varied between 2 to 4 sessions with 25% requiring up to 4 sessions and 50% requiring 2 sessions. Placebo or chemical introduction occurred in a randomized sequence post adaptation. Skin conductance, skin temperature, surface electromyography, heart rate and respiratory rate were used to measure response to challenge substances along with symptoms and environmental scores. Skin conductance seemed to be the obvious indicator of a response to the challenge substances. All patients reacted to the introduction of an antistatic fabric softener. 90% reacted to the glue and 80% reacted to the body wash solution in the patient group. While none of the controls reacted to the fabric softener or glue, one control reacted to the body wash solution. Symptom scores were higher for all substances in the patient group.

This study has now been confirmed in a formal study with a larger sample size showing a clear difference in the adaptation between the patients and the control group. The

reactivity to the challenge substance as indicated by the skin conductance is still higher in the patient group compared to the controls. The formal study has also revealed the complexity of the reactivity in individuals with MCS. The MCS patient reacts to the presence of chemical triggers, without conscious awareness as all the subjects wore nose plugs and could not smell the substance. Although there is correlation with symptoms, it is not possible to rely upon the conscious interpretation to establish an exposure. Further studies are planned to explore the role of conscious-unconscious awareness and correlation between physiological and symptomatic response.

Objectives & Notes

Dietrich K. Klinghardt, M.D., Ph.D. Date of talk: Saturday, June 11, 2005, 3:30pm

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Training:

Current Faculty Appointments:	Capitol University, Washington, DC
Medical School/University Attended:	Albert-Ludwig University Freiburg, Germany
Internship:	Albert Ludwig University, Freiburg, Germany
Residency:	Surgery University Clinic, Freiburg, Germany
Board Certifications:	Board certified in General Practice (Germany) and Pain Management, US

SPEECH TITLE: “The Sphenopalatine Ganglion and Environmental Sensitivity”

At the end of this Presentation, the participant should be able to:

1. Understand the anatomy and physiology of this ganglion.
2. Understand the pathophysiology of this ganglion: toxicity, infection, allergic ganglionitis, descending emotional influences, structural injury from poor occlusion and electric dysfunction from oral electrogalvanic currents (electrogalvanism).
3. Be aware of 3 techniques to restore health in this ganglion: injection, electric stimulation, psychoemotional techniques.

The Sphenopalatine Ganglion (SPG) and Environmental Sensitivity

Symptoms of MCS and related disorders often include chronic sinusitis, rhinitis, cognitive problems of the brain and inappropriate emotional states. Often there are related digestive problems, fatigue and the exaggerated reactions to inhalants, foods and odors. Besides the more known innervation of the saliva producing glands and the mucous producing cells of the sinus epithelium and tear glands, projections of the SPG have been found in the middle cerebral artery. The ganglion is mostly parasympathetic, but also has sympathetic connections. The superficial location in the pharynx explains the extraordinary sensitivity of this ganglion to odors and particles in the air. The neighborhood to the teeth suggests a vulnerability to mercury fumes escaping from dental amalgam fillings. The intricate connection to the vagus nerve and the innervation of the saliva producing glands predicts many of the digestive symptoms observed in dysfunctional states. The little explored access of the SPG to the deep cerebral arterial supply can explain many of the brain/limbic system related observations in MCS. Both the literature and clinical experience demonstrate that treating this ganglion can be a rewarding intervention in the treatment of environmental sensitivity.

Objectives & Notes

Jorge A. Ayala Moran, M.D.

Date of talk: Saturday, June 11, 2005,
4:00pm

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Medical School/ University Attended	Facultad de Medicina "Miguel Alemán" Universidad Veracruzana
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Residency:	Instituto Nacional de Pediatría, México Cytí
Board Certifications:	Consejo Mexicano de Otorrinolaringología, Soc. Mexicana de Alergia en ORL

Other Information:

Guia Clinica de Rinosinusitis, Academia Mexicana de Cirugia 2004

Are you looking the cause of disease?
Boletin de la Soc. Mexicana de Otorrinolaringology

SPEECH TITLE: “Allergy as a Cause of Sleep Apnea Syndrome”

At the end of this Presentation, the participant should be able to:

- 1.
- 2.
- 3.

The above information was provided by the Speaker.

Objectives & Notes

Kou Sakabe, M.D.

Date of talk: Saturday, June 11, 2005,
4:30pm

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Training:

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Board Certifications:	Japanese Society of Industrial and Occupational Medicine, Japanese Association of Physical Medicine, Balneology and Climatology

SPEECH TITLE: “Effect of EMF on Male Reproductive Organs”

At the end of this Presentation, the participant should be able to:

1. Understand the cellular effects of an extremely-low frequency magnetic field (EMF) on spermatogenesis.
2. Understand the endocrine disruptive effect of EMF on male reproductive organs.
3. Understand the effect of EMF on male reproductive organs for health risk assessment.

The above information was provided by the Speaker.

Effect of EMF on Male Reproductive Organs

Kou Sakabe, M.D., Ph.D.

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The cellular effects of an extremely-low-frequency electromagnetic field (EMF) on mouse spermatogenesis were assessed by DNA flow cytometry and serum testosterone. Seven week old male ICR mice were exposed to a 50 Hz EMF the strength of which was

1.0 m Tesla. Seven mice per treatment group were exposed for 13, 26, 39 or 52 days. For each experimental point, an equal number of mice per sham-treated group were used as a control and were exposed only to the background field below 1 μ Tesla in the same room as the treatment group.

In the control mice, the testis cellular DNA content distribution by flow cytometry was characterized by four quantifiable populations; round spermatids (1C), spermatogonia and other diploid cells (2C), spermatogonial cells synthesizing DNA (S-phase) and primary spermatocytes (4C).

In animals exposed for 26 days the number of cells in the 4C and the 4C:2C ratio was significantly lower, the 1C:4C ratio (meiotic transformation) was significantly higher than the corresponding control groups. In animals exposed for 52 days the cell population in 1C and the 1C:2C ratio (total germ-cell transformation) was significantly higher, and the cell population in 2C was significantly lower than the corresponding control groups.

The concentration of serum testosterone in animals exposed for 13 days was significantly higher than in the corresponding control group.

These changes suggest that long-term exposure to an extremely-low-frequency EMF had a possible effect on the proliferation and differentiation of spermatogonia. Moreover, the results of the present study suggest that we must recognize the possibility that EMF can affect the various testicular functions in the capacity of endocrine-disrupting factors.

Finally, the precise mechanism of EMF action on spermatogenesis is still unclear. Further efforts to resolve this question are underway in our laboratory.

Objectives & Notes

Aristo Vojdani, Ph.D.

Date of talk: Saturday, June 11, 2005,
5:00pm

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Training:

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Residency:	Post-Doctoral study at UCLA
Board Certifications:	Clinical Laboratory Scientist, State of California

CASE STUDY: "Neuroimmune Abnormalities in a Patient Exposed to a Combination of Mercury and Chemicals Used in Fumigation"

At the end of this Presentation, the participant should be able to:

1. Understand that chemicals can induce immune deficiency on the one hand, and autoimmunity on the other hand.
2. Observe that cellular immune abnormalities are detected side by side with humoral immune abnormalities in patients exposed to mercury and dursban.
3. Understand that these abnormalities cannot be detected by normal laboratory testing but can be documented by immunotoxicological and neurotoxicological evaluations.

The above information was provided by the Speaker.

Neuroimmune Abnormalities in a Patient Exposed to a Combination of Mercury and Chemicals Used in Fumigation.

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Abstract

A 53-year-old female practicing dentistry more than 20 years. Loved to eat fish, (mainly salmon), 2-4 times per week. Bought a new house and moved in immediately after fumigation. Three days later, she developed severe headache, dizziness, fatigue, and fibromyalgia.

After being unable to work for five days she decided to go to the emergency room. All physical exams and routine lab tests were found to be normal. During a period of 12 months she visited over 14 doctors; in most cases she was prescribed anti-depressants that did not ease symptoms.

After visiting 14 clinics the patient was referred to one of the AAEM members, who based on medical history and a physical exam, connected the patient's conditions to either mercury exposure or to Dursban used for fumigation. Blood samples were sent for immunotoxicological evaluations and the following abnormalities were detected: low % T-helper cells, high % T-suppressor cells, low T-helper/suppressor ratio, high % memory lymphocytes, low natural killer cell activity, and low lymphocyte response to T-cell mitogens and very high % of cells going through apoptosis. ANA titer was 1:640 with speckled pattern, elevated total immune complexes, elevated IgG antibodies against mercury bound to human serum albumin, fibrillarlin, chromatin, myelin basic protein, neurofilaments, and tubulin were detected.

These results indicated that the patient is having cellular immune deficiency and humoral immune activation resulting in autoimmunity. Analysis of the data may assist in finding mercury or Dursban as being responsible for the detection of immunodysregulation.

**23rd Annual International Symposium
on
Man and His Environment in Health and Disease**

**Special Focus
The Autonomic Nervous System and Its Relationship to Environmental
Pollutants Including the Cardiovascular System and Electromagnetic
Sensitivity**

**Sponsored by
American Environmental Health Foundation
and
University of North Texas Health Science Center**

Physician Accreditation/Credit:

This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education and the American Environmental Health Foundation. The University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education is accredited by the ACCME to provide continuing medical education for physicians.

The University of North Texas Health Science Center at Fort Worth is accredited by the American Osteopathic Association to award continuing medical education to physicians.

The University of North Texas Health Science Center at Fort Worth designates this educational activity for a maximum of 24 Category 1 credits toward the AMA Physician's Recognition Award. Each physician should claim only those credits that he/she actually spent in the activity.

The University of North Texas Health Science Center anticipates this program for 24 hours in Category 2A CME credit hours, pending approval from the American Osteopathic Association.

Nursing Accreditation/Credit:

University of North Texas Health Science Center at Fort Worth Office of Professional & Continuing Education, Provider #02588A, is approved provider of continuing nursing education by the Texas Nurses Association, an accredited approver by the American Nurses Credentialing Center's Commission on Accreditation. This activity

meets Type 1 criteria for mandatory continuing education requirements toward relicensure as established by the Board of Nurse Examiners for the State of Texas. This activity is approved for 28.8 Contact Hours.

To receive a certificate of successful completion, participants must attend the activity in its entirety and complete and return the activity evaluation credit request form.

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FINANCIAL CONSIDERATION

AEHF is a nonprofit organization that was founded in 1975 to provide education and research into Environmental Medicine. This year's Symposium is our 23rd Annual International Symposium and is our major vehicle for educating the medical professional.

Funding for the symposium is provided by registration fees from physicians and exhibitors. Proceeds from the AEHF store cover the shortfall between registration fees and expenses for the conference. AEHF does not receive grants or any outside financial support for our education. Donations are accepted and used toward research into environmental medicine.

INTRODUCTION

SYMPOSIUM PURPOSE

Since 1981, the International Symposium has been recognized as one of the most advanced medical forums in the world addressing the research and treatment of environmental effects on health and disease. The 2005 conference will focus on "The Autonomic Nervous System and Its Relationship to Environmental Pollutants Including the Cardiovascular System and Electromagnetic Sensitivity". This Conference presents the most current information available while providing guidelines to identify, diagnose, treat and to prevent environmentally triggered responses in the body.

GOALS OF THE MEETING

- ! To provide new insights in *The Autonomic Nervous System and Its Relationship to Environmental Pollutants* and the environmental causes behind many problems you see.
- ! To present new diagnostic and treatment modalities to help you improve the quality of care for your complex patients.
- ! To provide concepts, tools that will enhance your practice.

OBJECTIVES OF THE MEETING

- ! Improve the outcome of treating patients with sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Use new concepts and treatments to help better diagnose and manage many patients with environmentally triggered problems and sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Apply the concepts of this conference to your practice by using nutrition and environmental manipulation for the treatment of sensitivities to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.
- ! Use the information presented to enhance the effectiveness, cost-efficiency, and competitiveness of the physician in relation to *The Autonomic Nervous System and Its Relationship to Environmental Pollutants*.

INTENDED AUDIENCE

M.D.=s, D.O.=s, medical students, nurses, nutritionists and other health professionals interested in the concepts and practice of Environmental Medicine, Occupational Medicine and Toxicology.

EDUCATIONAL FORMATS

- ✂ Plenary
- ✂ Panels Discussions
- ✂ Case Studies
- ✂ Question & Answer Sessions.

CONFERENCE FORMAT

The AEHF Committee has selected some of the leading experts in the fields of chronic disease, nutrition and chemical sensitivity.

Each speaker=s presentation will last approximately 20 minutes and will be followed by a 10 minute question and answer session. All speakers are encouraged to use any and all appropriate audio/visual aids. (A brief outline of the speech is included in this booklet.)

GIVEN IN COOPERATION

William J. Rea, M.D., F.A.C.S.

Symposium Chairman,
American Environmental Health Foundation,
Environmental Health Center - Dallas,
Dallas, Texas

Bertie B. Griffiths, Ph.D.,

Environmental Health Center - Dallas
Dallas, Texas

Kaye H. Kilburn, M. D.

University of Southern California Medical Center
Keck School of Medicine
Los Angeles, CA

William J. Meggs, M.D., Ph.D.

Brody School of Medicine, East Carolina University
Department of Emergency Medicine
Greenville, NC

23rd Annual International Symposium on Man and His Environment

Schedule

Sunday, June 12, 2005

8:30 **ANNOUNCEMENTS/MODERATOR:** Doug Seba Ph.D

9:05 “Interactions Between Immune and Autonomic Systems in Patients Sensitive to foods & Chemicals”,
Colin Little M.D.

9:25 Q & A

9:35 “Organic Markets – from Grains and Oilseeds to Fibers”, Lynn Clarkson

9:55 Q & A

10:05 **MORNING BREAK WITH EXHIBITORS**

10:30 “Environmental Hydrogen Sulfide: Rethinking Acceptable Exposure Levels”,
William J. Meggs, M.D., Ph.D.

10:50 Q & A

11:00 “ANS Involvement in Chemical and Electromagnetic Sensitivities”, Cyril Smith, Ph.D.

11:20 Q & A

11:30 “Stress Augments Chemical - Induced Neurotoxicity”, Mohamed B. Abou-Donia, Ph.D.

11:50 Q & A

12:00 **SUMMARY AND CLOSE:** Doug Seba Ph.D

SUNDAY, JUNE 12, 2005

ABSTRACTS

AND

HANDOUTS

Objectives & Notes

Colin Little, M.D.

Date of talk: Sunday, June 12, 2005, 9:05am

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Mt. Waverley
Melbourne, Victoria 3149
Australia

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Training:

Current Job Description:	Physician and Allergist, Research participant in CSIRO project on cow's milk intolerance in adults.
Medical School/University Attended:	Melbourne University
Internship:	Western General Hospital
Residency:	Western General Hospital
Board Certifications:	MB, BS, MRCP (U.K.), FRACP, FACA
Other Information:	Current trends in Immunology Vol3 (2000) pgs131-144; Proceedings Soc Exp Biol Med Vol 224 (2000) pgs 264-272; Exp Biol Med (Mini review) Vol227 (2002) pages 438-444; Infect Immun Vol 68 (2000) pgs 3840-47

SPEECH TITLE: **“Interactions Between Immune and Autonomic Systems in Patients Sensitive to Foods and Chemicals”**

At the end of this Presentation, the participant should be able to:

1. Understand connections between immunity and sensory nerves.
2. Interactions between sensory nerves and brain centers (incl. ANS)
3. Application of this knowledge to examples of food intolerance and chemical sensitivity.

The above information was provided by the Speaker

INTERACTIONS BETWEEN IMMUNE AND AUTONOMIC SYSTEMS IN PATIENTS SENSITIVE TO FOODS AND CHEMICALS.

There is limited knowledge of the interaction between the immune and autonomic nervous systems. This discussion focuses on sources of afferent stimuli in patients with sensitivity disorders and discusses the current data on autonomic function in clinical conditions where food or chemical sensitivity may be important.

Studies over the past decade have demonstrated that immune stimuli signal the central nervous system not only by the humoral route but via sensory fibres, particularly c fibres relaying pain sensation. The vagus nerve is the principal conveyor of these neural stimuli but the glossopharyngeal and trigeminal nerves almost certainly have a similar function. This sensory information is relayed to the brain stem, particularly the Nucleus Tractus Solitarius, amygdala and hypothalamus. Complex interactions occur with autonomic centers with (potentially) variable autonomic responses. Such responses may influence the function of the gut, respiratory tract and other sites such as the heart and skin. Other concurrent influences on the central nervous system will further modulate these responses.

There is increasing evidence for activation of the sensory nerves in patients with food intolerance associated with symptoms of an Irritable Bowel Syndrome. Patients with this disorder have a heightened responsiveness of the gut sensory nerves, increased mast cell numbers in the gut wall, evidence for local mast cell degranulation and close apposition between sensory nerve endings and mast cells. In children with cow's milk intolerance, studies have shown increased levels of the cytokine TNF α in the gut wall and in fecal fluid. This cytokine is a potent activator of c fibres.

There is also evidence that patients with chemical sensitivity show a heightened responsiveness of sensory nerves of the respiratory tract. One recent study demonstrated the release of neuropeptides from sensory nerves following chemical exposure. Such changes were not seen on a control group. Studies by Millqvist have demonstrated that chemically sensitive subjects have a lower threshold of the cough response to capsaicin. Increased levels of the cytokine TNF α have been detected in the sputum of patients with idiopathic cough and such patients also show increased levels of substance P in nasal lavage specimens. Finally, chemically sensitive patients have increased NGF in nasal secretions after capsaicin inhalation.

A study by a Dutch group may provide insight as to how chemicals may cause a heightened responsiveness of sensory nerves. They sensitized mice with dinitrofluorobenzene. A subsequent challenge with dinitrobenzene sulphonic acid released TNF α into the tracheal fluid within fifteen minutes. This TNF α increased the susceptibility of sensory neurones to both the chemical (dinitrobenzene sulphonic acid) and also capsaicin. Antibodies to TNF α and TNF α receptor inhibited these effects. The process was shown to be mast cell dependent. Subsequent communication indicates that the immune response to the chemical is mediated by TH1 cells and that kappa light chains may be involved.

As mentioned, there are complex interactions between nuclei in the brain stem and centers mediating sympathetic and parasympathetic responses. For this reason stereotyped responses by the autonomic nervous system are unlikely to occur and will be contingent on other influences – stress, sleep-waking cycles, hormonal fluctuations etc. Data on the effects of cytokines released in peripheral tissues on local autonomic function is limited and also probably influenced by multiple parameters.

In general the peripheral release of cytokines such as IL-1 and TNF α activate the sympathetic nervous system with the release of noradrenaline, particularly in lymphoid organs. Immunological stimuli affecting the gut induces the release of acetylcholine from efferent parasympathetic fibres. This mediator inhibits cytokine release by macrophages and is thought to be protective against shock. In some tissues, for example the jejunum and atria, IL-1 and TNF α inhibit noradrenaline release. The general view to date is that the response of organisms innervated by the autonomic nervous system varies in response to the type of immune activation.

Although no firm conclusions can be drawn, it is useful to list the findings on autonomic function in a number of clinical conditions where there may be underlying sensitivity to foods and chemicals. They include vasomotor rhinitis, irritable bowel syndrome, migraine and chronic fatigue syndrome.

The data on autonomic function in vasomotor rhinitis is of considerable interest. Studies show a hypoactive sympathetic response. The situation is less clear cut in the irritable bowel syndrome. There are reports of increased parasympathetic and reduced sympathetic activity in patients with increased intestinal motility. The converse may apply in patients with decreased motility. There may be “dysregulation” of autonomic function but no clear cut pattern. In migraine sympathetic activity appears to be reduced with depletion of noradrenaline stores, at least in the pupil. In the chronic fatigue

syndrome there is again evidence of abnormal autonomic function without a consistent pattern. The tentative view is that the autonomic system is dysregulated.

In these conditions there may be efferent signaling, derived from immune responses, at least in some cases. The subsequent effects on autonomic function may depend on not only this signaling but on other concurrent influences on brain centers affecting autonomic function.

Objectives & Notes

Lynn Clarkson

Date of talk: Sunday, June 12, 2005, 9:35am

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Training:

Current Job Description:	President of Clarkson Grain Company
Medical School/ University Attended	University of Wisconsin, Stanford, Knox, Tulane

SPEECH TITLE: “Organic Markets – from Grains and Oilseeds to Fibers”

At the end of this Presentation, the participant should be able to:

1. Understand the scope of organic agriculture.
2. Understand the organic market price structure.
3. Have a perspective on the trends in organic food and fiber.

The above information was provided by the Speaker.

Abstract:

Title: *Organic Markets - from grains and oilseeds to fibers*

Presentation to the American Environmental Health Foundation's 23rd Annual Int'l Symposium

By: Lynn Clarkson

Organic farming is the brightest spot in US agriculture. Demand has been growing by over 20% per year since 1991 and should reach roughly \$20 billion by the end of this year. About 60 million Americans regularly pay premiums to get the organic foods they prefer raised in a way they support. That demand translates into price premiums of over 100% at the farm gate. Despite those incentives, domestic production is not keeping up with this burgeoning demand. Foreign supplies are stabilizing the market as more domestic farmers ponder their options. This presentation walks you through today's organic marketplace from farm to grocery store and consumer decision. For those who want greater care for the environment, fewer pesticides and better flavor, there is cause for celebration

Objectives & Notes

William J. Meggs, M.D., Ph.D.

Date of talk: Sunday, June 12, 2005, 10:30 am

Brody School of Medicine, East
Carolina University
600 Moye Blvd.
PCMH, 3ED-311, Department of
Emergency Medicine
Greenville, NC 27834-4354

Phone: 252/744-2954
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Training:

Current Job Description:	Medical School Professor
Current Faculty Appointments:	Allergy & Immunology Brody School of Medicine
Medical School/University Attended:	University of Miami School of Medicine
Internship:	University of Rochester
Residency:	University of Rochester; Fellowships: NIH, NYU
Board Certifications:	Medical Toxicology, Internal Medicine, Emergency Medicine
Other Information:	Co-author of "The Inflammation Cure", Scientific Progress in Understanding Gulf War Illnesses.

SPEECH TITLE: "Environmental Hydrogen Sulfide: Rethinking Acceptable Exposure Levels"

At the end of this Presentation, the participant should be able to:

1. To know the toxicity of acute & chronic exposures to hydrogen sulfide
2. To know the toxicity experienced in communities living near industrial facilities releasing hydrogen sulfide.
3. To know changes in regulatory status of hydrogen sulfide.

The above information was provided by the Speaker.

Environmental Hydrogen Sulfide: Rethinking Acceptable Exposure Levels

William Joel Meggs, MD, PhD
Brody School of Medicine
East Carolina University
Greenville, NC
USA

Hydrogen sulfide is a colorless gas that is heavier than air. It is extremely toxic as an irritant gas, cellular toxin, and has a noxious odor. Communities are exposed to hydrogen sulfide from pulp plants, refineries, decay of organ matter and composting of manure, slaughter houses and tanneries, and from volcanic activity. Community levels can range from tens to hundreds of parts per billion. Textbook authors and regulating bodies have considered toxic levels to be above a part per million. Several studies of communities have shown a high level of disease in persons exposed to hydrogen sulfide in levels traditionally considered safe. The experience with hydrogen sulfide points out the fallacies associated with setting safe limits for toxic substances.

Objectives & Notes

Cyril Smith, Ph.D.

Date of talk: Sunday, June 12, 2005,
11:00am

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U.K.

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Training:

Current Job Description:

Retired

Medical School/University Attended:

Imperial College, University of London,
England

Other Information:

Smith C.W. (2002) *Effects of Electromagnetic Fields in the Living Environment*. Proc. Intl. Conf. Electromagnetic Environments and Health in Buildings, Royal College of Physicians,

London, 16-17 May, 2002. In: Clements-Croome D (Ed.). *Electromagnetic Environments and Health in Buildings*. London: Taylor & Francis, October 2003. Chap. 3, pp. 53-118. ISBN 0-415-316-561

Smith CW. (2003) Guest Editorial – Straws in the Wind. *Journal Alternative and Complementary Medicine* **9(1)**: 1-6.

Smith CW. (2003) Guest Editorial – Energy Medicine United. *Complementary Therapies in Nursing and Midwifery* **9(4)**:169-175 (Nov 2003).

Smith CW. (2004) Quanta and Coherence Effects in Water and Living Systems. *J Altern Complement Med.* 10(1);69-78.

SPEECH TITLE: “ANS Involvement in Chemical and Electromagnetic Sensitivities”

At the end of this Presentation, the participant should be able to:

1. Appreciate the duality between frequency and chemical structure
2. Understand the ANS interactions and ANS stability
3. Appreciate the effects of chemicals and electromagnetic fields, acupuncture, and homoeopathy on the ANS.

The above information was provided by the Speaker.

**23rd ANNUAL INTERNATIONAL SYMPOSIUM
ON MAN & HIS ENVIRONMENT**

Abstract

**“Electromagnetic Sensitivity and the ANS”
and
“ANS Involvement in Chemical and Electromagnetic Sensitivities”
Cyril Smith, Ph.D.**

The first part of these two presentations is intended to enable the participant to recognize Electromagnetic Sensitivities in patients, to appreciate the importance of endogenous and exogenous frequencies and the relationship between these frequencies and the autonomic nervous system.

The second part is intended to explain the duality between frequencies and chemical structure and their relation to autonomic nervous system stability criteria and interactions in living systems. The effects of interactions between chemical in the body and environmental electromagnetic fields is explained in terms of the endogenous frequencies on acupuncture meridians and linkages between specific acupuncture points and points in the autonomic nervous system.

The conclusions are that Voll's link between these specific acupuncture points and the ANS gives an insight into how environmental frequencies can interact with the ANS.

The Multiple Frequency Effect in coherent systems shows how the frequency signatures of chemicals can behave like environmental frequencies and vice versa.

References to the writer's publications on “Electrical Hypersensitivity and Water Phenomena”, and previous presentations at these International Annual Symposia on “Man and His Environment in Health and Disease” held in Dallas, Texas, from 1986-2000 are listed in the Handout which also includes some definitions of electromagnetic quantities.

A set of “Notes for Patients on Electrical Sensitivities” written for the Breakspear hospital, England are also appended as these may be found useful for clinicians to have available.

(They are listed in the Friday section of your syllabus at 9:35 am.)

Objectives & Notes

Mohamed B. Abou-Donia, Ph.D.

Date of talk: Sunday, June 12, 2005,
11:30am

Duke University Medical Center

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Laboratory of Neurotoxicology

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Dept. of Pharmacology and Cancer

Email: donia@acpub.duke.edu

Biology, Box 3813

Durham, NC 27710

Training:

Current Job Description:

Professor of Pharmacology and Cancer
Biology

Current Faculty Appointments:

Duke University Medical Center

Medical School/University Attended:

University of California, Berkeley

Internship:

N/A

Residency:

N/A

Board Certifications:

American Board of Toxicology (ABT), and
Academy of Toxicological Sciences (ATS)

Other Information:

Book Editor, *Neurotoxicology*, CRC;
Publications: more than 300.

SPEECH TITLE: "Stress Augments Chemical - Induced Neurotoxicity"

At the end of this Presentation, the participant should be able to:

1. Know that exposure to stress alone results in neuronal cell damage.
2. Realize that stress increases chemical-induced neurotoxicity.
3. Both chemicals and stress induce neuronal injury via oxidative stress by increasing neuronal reactive oxygen species.

The above information was provided by the Speaker.

Stress Augments Neurological Deficits Induced by Combined Exposure to Pyridostigmine Bromide, DEET, and Permethrin. Mohamed B. Abou-Donia, Ashok Shetty, and Ali A. Abdel-Rahman, Department of Pharmacology and Cancer Biology, Duke University Medical center, Durham, North Carolina

Exposure to a combination of stress and low doses of chemicals pyridostigmine bromide (PB), DEET and permethrin in adult rats, a model of Gulf War exposure, produces blood-brain-barrier (BBB) disruption and neuronal cell death in the cingulate cortex, dentate gyrus, thalamus and the hypothalamus. In this study, neuropathological alterations in other areas of the brain where no apparent BBB disruption was observed was studied following the above exposure. Animals exposed to both stress and chemicals exhibited decreased brain AChE activity in the midbrain, brainstem and the cerebellum and a decreased m2 muscarinic ACh receptor ligand binding in the midbrain and the cerebellum. These alterations were associated with significant neuronal cell death, reduced MAP-2 expression, and increased GFAP expression in the cerebral cortex and the hippocampal subfields CA1 and CA3. In the cerebellum, the neurochemical alterations were associated with Purkinje cell loss and increased GFAP immunoreactivity in the white matter. However, animals subjected to either stress or chemicals alone did not show any of the above changes in comparison to vehicle treated controls. Collectively, the above results suggest that prolonged exposure to a combination of stress and the chemicals PB, DEET and permethrin can produce significant damage to the cerebral cortex, hippocampus and the cerebellum, even in the absence of apparent BBB damage. As these areas of the brain are respectively important for the maintenance of motor and sensory functions, learning and memory, and gait and coordination of movements, the above alterations could lead to many physiological, pharmacological, and behavioral abnormalities, particularly motor deficits and learning and memory dysfunction. (Supported in part by the U.S. Army Medical Research and Materiel Command under contract project order DAMD 17-99-1-9020).