18th Annual International Symposium

On

Man and His Environment

June 8-11, 2000

Dallas, Texas

Introduction

Welcome to the 18th Annual International Symposium on Man and His Environment in Health and Disease. This Symposium is one of the most advanced forums in the world addressing the research and treatment of environmental effects on health and disease.

At this year's Conference, experts from throughout the world will share their extensive experience and specialized knowledge with an audience of physicians, scientists and health professionals.

Special Focus

The 2000 Annual International Symposium will focus on the Environmental Aspects of Cardiovascular Disease. This Conference will explore some of the latest data and provide a forum for discussion as well as case studies to help the professional.

Goals of this Symposium

- To provide important new insights into the mechanisms, and the environmental causes behind many problems seen in your practice.
- To present new diagnostic and treatment modalities to help you improve the quality of care for your patients.
- To provide concepts and tools that will enhance your practice.

Objectives of the Symposium

- Improve the outcome of treating cardiovascular disease.
- Use new concepts and treatments to help better diagnose and manage many patients with chemical sensitivity.
- Apply the concepts of a longevity medicine to your practice.

- Use the information presented to enhance the effectiveness, cost-efficiency, and competitiveness of your practice.

Conference Format

The AEHF Committee has selected some of the leading experts in the field of Environmental Aspects of Cardiovascular Disease.

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.)

Every afternoon, we will have a case study/panel discussion. This session will consist of various faculty members discussing real cases. The audience is encouraged to participate in these discussions.

Accreditation

This activity has been planned and implemented in accordance -with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of the American Academy of Environmental Medicine (AAEM) and the American Environmental Health Foundation. The American Academy of Environmental Medicine is accredited by the ACCME to provide continuing medical education for physicians.

The American Academy of Environmental Medicine designates this educational activity for a maximum of 22 hours in Category 1 credits towards the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

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 Griffiths, Ph.D.,
Environmental Health Center – Dallas
Dallas, Texas

Ervin J. Fenyves, Ph.D.,
Department of Physics and Environmental Sciences

University of Texas at Dallas
Richardson, Texas

Nancy A. Didriksen, Ph.D.,
Health Psychology/Behavioral Medicine
Richardson, Texas

Supporters of the

Annual International Symposium

A Special thanks to all of the companies listed below that have continually supported our Annual International Symposium for more than ten years.

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- Klaire Laboratories Inc.

SCHEDULED FACULTY, SPEECH TITLES AND BIOGRAPHIES

Malcolm Beck Phone: 210/651-6115
7561 East Evans Rd. Fax: 210/651-9231
San Antonio, TX 78266

Title: "The Secret Life of Compost" and "Understanding the Insect/Pest"

Life time student of nature, owned and operated two organic farms producing vegetables, fruit, pecans and some farm animals since 1957. Founder of Garden-Ville Fertilizer Co., the headquarters for the organic gardener. Authored books, many pamphlets and articles, co-authored two books, give presentations from 50 to 70 times each year to Colleges & Universities.

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Tonya G. Callaway Phone: 972-724-0090
5028 Timbercreek Rd. E-mail:tgc0005@unt.edu
Flower Mound, TX 75028

Current Job: University of North Texas, Dept. of Rehabilitation, Social Work, & Addictions, Neurotherapy Lab. Providing biofeedback to clients. Tonya G. Callaway, M.S. Currently a doctoral student at the University of North Texas, M.S. Currently a doctoral student at the University of North Texas, Health Psychology/Behavioral Medicine Department. Working on QEEG research with Dr. Dan Miller, Chair of Psychology Department Texas Women's University. Also working on research with neurotoxically-exposed patients under the supervision of Dr. Nancy Didriksen.

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Thomas Croley, Ph.D. Phone: 214/368-4132
Environmental Health Center - Dallas Fax: 214/691-8432
8345 Walnut Hill Lane, Suite 220
Dallas, TX 75231

Title: "Effective Use of Cold Laser Therapy In The Treatment of Environmentally Challenged Patients"

Education: Ph.D. from Baylor college of Dentistry in Human Anatomy in 1971. Post Doctoral Education: NASA-Manned Spacecraft Center-"Effects of Lunar Dust on Tissue Cultured Cells", Paraprofessional Education: Diagnostic Cytologist (A.S.C.P.) Southwestern medical School, Diagnostic Microbiologist for Wadley Blood Center. Teaching Experience: 30 years in all disciplines of anatomy and all allied health professionals. 97 individual thesis/dissertation student committees. Elected Board of Advisors of International Academy of Scientific Acupuncture 1999.

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Nancy Didricksen, Ph.D. Phone: 972/889-9933
100 N. Cottonwood Dr., Suite 106 Fax: 972/889-9935
Richardson TX 75080

Title: "The Use of the Halstead-Reitan Neuropsychological Test Battery to Measure Neurotoxic Effects in Chemically-Exposed Individuals"

- Ph.D. in Health Psychology/Behavior Medicine from University of North Texas with research in psychoneuroimmunology.
- Approximately fifteen years experience evaluating and treating environmentally ill patients in both in and out- patient settings as well as other chronically ill patients.
- Currently in private practice in Richardson Texas evaluating and treating environmentally ill patients.
- Adjunct Professor of Psychology at University of North Texas with research emphasis on the adverse neuropsychological effects of environmental illness/chemical sensitivity.
- Approximately 40 professional papers and presentations.

Howard Garrett Phone: 817/695-0817
The Natural Way Fax: 214/365-0608
P.O. Box 140650 E-mail:hgarrett@wbap.com
Dallas, TX 75214

Titles: "Creating the Edible Landscape" and "Growing Ornamentals and Food Crops the Natural Way"

Landscape architect, columnist for Dallas Morning News, Gardening talk show host on WBAP-820, author of seven books on gardening, landscaping and pest control. Consultant on farming, ranching and landscaping.

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Bertie Griffiths, Ph.D. Phone: 214/368-4132
Environmental Health Center - Dallas Fax: 214/691-8432
8345 Walnut Hill Lane, Suite 220
Dallas, TX 75231

Title: "Response of the Cell Cycle to Nutrition"

Graduate of the University of Wisconsin and University of the West Indies, Faculty of medicine. Recipient of Degrees in microbiology, virology, and postdoctoral training in Infectious Diseases and Immunology. Rockefeller fellowship to study Entomology and Virus epidemiology in Brazil and Trinidad. Appointments: Professor and Consultant in Microbiology and Infectious Diseases. Presently Director of EHC-D Laboratory.

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Satoshi Ishikawa, M.D. Phone: 81/42-795-5784
Kitasato University Fax: 81/42-799-2287
Director Environmental Health E-mail:ishikawa@kitasato-u.ac.jp
4-5-19 Minami-tsukushino
Machida, Tokyo 194-0002 Japan

Title: "Chemical Sensitivity Patients in Japan, Part I" and " Results of visuo-oculomotor functions of Chemical Sensitivity, Part II"

Fulbright Scholarship to NYU Medical Center, Neuro-ophthalmology & toxicology, Professor and chairman, Kitasato University, School of Medicine, Fellow of American Academy of Environmental Medicine, Dean, Kitasato University, School of Medicine, Jonathan Forman Award at Boston 31st Meeting, Director of Clinical Environmental Medical Center, Kitasato Institute

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Kaye Kilburn, M. D. Phone: 323/442-1830
University of Southern California Fax: 323/442-1833
Keck School of Medicine E-mail:kilburn@hsc.usc.edu
2025 Zonal Ave., CSC-201
Los Angeles, CA 90033

Titles: "Heart Rate Variation in Adults Exposed to Chlorine and Creosol" and "A Coming Catastrophe: Tobacco and Myocardial Infarction"

Kaye H. Kilburn, M.D. has spent 50 years in medicine and holds the Edgington Chair at University of Southern California, Keck School of Medicine. Cardiology, pulmonary disease and brain impairment have had his attention. The author of "Chemical Brain Injury" (John Wiley and Sons, 1998). Published 250 scientific papers, many dealing with chemical effects on the lungs and nearly 40 concerning chemical effects of the brain. Using sensitive tests he has assessed effects of chlorine, hydrogen sulfide, chlorpyrifos (Dursban), chlordane, pyrethroids (permethrin), ammonia, arsenic and diesel exhaust in more than 3,500 people.

Rima E. Laibow, M.D. Phone: 914-827-9557
Medical Director Fax: 914-827-3995
Alexandria Institute of Natural and Integrative Medicine E-mail:laibow@juno.com
10 Old Post Road South
Croton on Hudson, NY 10520

Title: "Cardiac parameters and finger temperature response to Q-EEG-Neuro BioFeedback rehabilitation of brain-injured patients."

Dr. Laibow is the medical director of the Alexandria Institute of Natural and Integrative Medicine, Croton, NY. She is world renowned for her innovative use of Neuro Bio Feedback in the treatment of supposedly "incurable" diseases, injuries and conditions. Her practice integrates nontoxic modalities with non invasive pharmaceutical-free therapies. Dr. Laibow is a graduate of Albert Einstein College of Medicine.

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Stephen Levine, Ph.D. Phone: 415/453-0478
NutriCology, Inc. Fax: 415/925-1356
15 Bridge Rd. E-mail:salevine@ix.netcom.com
Kentfield, CA 94904

Title: "Antioxidant Adaptation, Cancer and Cardiovascular Disease"

Received N.H. full fellowship, trained at U.C. Berkeley in molecular biology and genetics. Received Ph.D. in 1976. Functioned as consultant in nutrition until 1980, then started Allergy Research Group. Published many technical and regular newsletters and lectured to professionals. In 1984 published a classic text "Antioxidant Adaptation: Its Role in free Radical Biochemistry", co authored by Parris Kidd, Ph.D. Since then has researched biological process from energetic perspective of free radical biochemistry.

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Allan Lieberman, M.D. Phone: 843/572-1600
7510 Northforest Dr. Fax: 843/572-1795
North Charleston, SC 29420

Title: "Pesticide Poisoning - The Missed Diagnosis"

Board certified in Environmental Medicine since 1988, Assistant Professor - Biochemistry at Brown University, Director of Center for Occupational Environmental Medicine, N. Charleston, South Carolina, Consultant in Clinical Research - MILKHAUS LABS. Special Interests: Biodetoxification, Organophosphate, Pesticide Toxicity

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William Meggs, M.D. Phone: 252/816-2954
E. Carolina University School of Medicine Fax: 252/816-3589
Dept. of Emergency Medicine E-mail:meggs@brody.med.ecu.edu
Brody Bldg., Room 4W54
Greenville, NC 27858

Titles: "Allergy and the Heart" and "Toxins and the Heart"

Dr. William Meggs is a Professor at East Caroline University, where he is Chief of Toxicology. Dr. Meggs is board certified in Medical Toxicology, Allergy & Immunology, Internal Medicine, and Emergency Medicine. He is a graduate of the University of Miami School of Medicine and was a medical staff Fellow at NIH. Research interests include chemical sensitivities, organophosphate poisoning, and quantum biology.

Jean Monro, M.D. Phone: 011/44-1441-261333
Breakspear Hospital Fax: 011/44-1442-266388
Belswains Lane E-mail:nmonr@breakspear.org
Hemel Hempstead, Herts HP3 9HP, England

Titles: "Heart Rate Variability in the Food and Chemically Sensitive Patient" and " Electromagnetic Screening as a Protection from telephone Frequencies and Effects on Heart Rate Variability."

Dr. Monro attended London Hospital Medical School, England. Her residency was at London Hospital. Her board certifications are MB, B.S., MRCS, LRCP, FAAEM, DIBEM, and MACOEM. Her current job description is she is the Medical Director of Breakspear Hospital, England.

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Prof. Garth L. Nicolson Phone: 714/903-2900
Nancy L. Nicolson Fax: 714/379-2082
The Institute for Molecular Medicine E-mail:gnicimm@ix.netcom.com
15162 Triton Lane
Huntington Beach, CA 92649-1041

Title: "Diagnosis and Treatment of Cell-Invasive Bacterial Infections in CFS, FMS, Gulf War Illness and Rheumatoid Arthritis"

Currently Professor of Internal Medicine (Research) and President and Chief Scientific Officer of the Institute for Molecular Medicine, Hunting Beach, California. Dr. Nicolson has published over 500 medical/scientific papers, editor of two journals and associate editor of twelve journals. Graduate of University of California in San Diego.

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Jon Pangborn, Ph.D. Phone: 630/587-4458
Bionostics, Inc. Fax: 630/587-4465
42W719 Bridle Court
St. Charles, IL 60175

Titles: "A Clinical Assessment of Taurine, a Modulator of Cellular Electrolytes" and "Update on Clinical Aspects of Homocysteine"

Dr. Jon Pangborn is President of Bionostics, Inc., which consults with physicians, clinics and clinical laboratories on laboratory testing and on nutritional remedies for metabolism and toxicity problems. Dr. Pangborn also does research in the area of childhood development disorders and autism. He is a Fellow of the American Institute of Chemists, and is Adjunct Professor of Nutritional Biochemistry at the Union Institute.

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Kalpanna Patel, M.D. Phone: 716/837-1320
Northwest Center for Allergy & Environmental Medicine Fax: 716/833-2244
65 Wehrle Dr. E-mail: aehcwhy@wny
Buffalo NY 14225

Title: "Effectiveness of Estrogen replacement and Intravenous Magnesium Therapy to Correct Dyshomeostasis and Dysfunctioning Vascular Endothelium"

Diplomate of American Board of Pediatrics and American Board of Environmental Medicine. She is President of the American and International Board of Environmental Medicine and is the Director of the Allergy and Environmental Health Center in Buffalo, New York. She is in private practice of Environmental Medicine for the last 18 years.

William J. Rea, M.D. Phone: 214/368-4132
Environmental Health Center - Dallas Fax: 214/691-8432
8345 Walnut Hill Lane, Suite 220 E-mail:wjr@ehcd.com
Dallas, TX 75231

Titles: "The Environmental Aspects of Cardiovascular Disease, Part I" and "The Environmental Aspects of Cardiovascular Disease, Part II"

Graduated with an M.D. from Ohio State University College of Medicine in 1962. Board Certified in Environmental Medicine, Thoracic and General Surgery. Member of the AAEM, Pan Am Allergy Society, American Academy of environmental Otolaryngic Allergy. Received the Jonathan Forman Fold Medal award and the Herbert J. Rinkel Award from the AAEM. Books: The author of Chemical Sensitivity, Volumes I, II, III, and IV; Your Home, Your Health, and Your Well Being, and Success in a Clean Bedroom.

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Alexander Riftine, Ph.D. Phone: 732/635-9100
173 Essex Ave. Fax: 932/635-1144
Metuchen, NJ 08840 E-mail:hrinstr@hotmail.com

Title: "Heart Variability In Russian Sailors"

Dr. Riftine received his Ph.D. in Biological Sciences from Glushkov Institute of Cybernetics, Kiev, USSR, in 1987. He received an M.S. in Automation and Computer Engineering, from Leningrad Naval Academy, in 1972. He is currently the President and Scientific Director of Heart Rhythm Instruments, Inc. His experiences include Laboratory-based physiological research including computer analysis of the functional state of physiological systems based on analysis of Heart Rate Variability. Automated diagnosis research for cardiovascular, pulmonary, and other functional systems.

Development of computer methodologies and software for physiological research, including artificial intelligence techniques and mathematical analysis of random processes. Development of Health-Express, an automated computer-based health monitoring system for the evaluation of the functional state of the autonomic nervous system.

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Sherry Rogers, M.D. Phone: 315/488-2856
Northeast Center for Environmental Medicine Fax: 315/488-7518
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Syracuse, NY 13219

Titles: "The Environmental Medicine Approach to Solving Recalcitrant Cardiology Conditions" and "Mechanisms of How Current Cardiology Therapies Eventually Exacerbate Symptoms as Well as Create New Diseases"

Sherry A. Rogers, MD, in private practice for 30 years, is a diplomate of the American Board of Family Practice and American board of Environmental Medicine and a Fellow of the American College of Allergy, Asthma, Immunology. She has published over a dozen books, a referenced newsletter for 10 years, 18 scientific papers and chapters in textbooks, and more.

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Kou Sakabe, M.D. Phone: 81/3-5490-2366
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Tokyo, Japan 108-8642

Title: "Environmental Signals and Immune Response"

Tokai University School of Medicine, Japan 1982 MD

Tokai University School of Medicine, Research Associate 1982-1988 Cell Biol.

Tufts University School of Medicine, Boston, Research Associate 1988-1990 Cell Biol.

Tokai University School of Medicine, Assist. Professor 1990-1994 Cell Biol.

Tokai University School of Medicine, Associate Professor 1997-1999 Cell Biol.

Kitasato Institute Hospital, Environmental Medical Center 2000- present

Doug Seba, Ph.D. Phone: 703/949-1055
P.O. Box 1417, #323
Alexandria, VA 22313

Titles: "Global Environmental Update 2000" and "Endocrine Disruptors in the Bitterroots"

Dr. Seba has over forty years experience in chemicals and health effects. He has a Ph.D. in Environmental Oceanography from the University of Miami. He is widely published in toxicology, ecology and information sciences. His current interest is in the fate and transport of endocrine disruptors.

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Theodore R. Simon, M.D. Phone: 214/528-2482
Nuclear Medicine Consultants of Texas Fax: 972/566-4762
4429 Southern Ave.
Dallas, TX 75202

Title: "Cardiovascular Nuclear Scanning"

1988-1990 Deputy Chief, Nuclear Medicine Service, National Institutes of Health

1980-present Associate Professor, Clinical Radiology, University Texas Southwest Medical School

1990-present Private Practice, Nuclear Medicine

1975 Yale University School of Medicine, MD

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Cyril W. Smith, Ph.D. Phone: 011/44-161-789-4768
Honorary Senior Lecturer (Retired), Fax(@ Salford University):

School of Acoustics and Electronics, 011/44-161-295-5145
University of Salford, E-mail:cyril.smith@which.net
Salford M5 4WT, England

Titles: "The Diagnosis and Therapy of EM Hypersensitivity" and "EM Fields in Health, in Therapies, as Hazards"

Since 1947 involved in electronics, physics research and teaching; PhD on X-ray images, Imperial College, London (1964). From 1970's at Salford University, EM properties bio-molecules, living systems, water. Since 1982, diagnosis / therapy of EM hypersensitivity. Presentations at these Symposia since 1986. Over 100 research publications, co-author "Electromagnetic Man" (1989).

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V.A. New Jersey Health Care System Fax: 973/678-2242
385 Tremont Ave. E-mail:wedeen@umdnj.edu
East Orange, NJ 07018-1095

Titles: "Lead and Hypertension: Who Cares?" and "Occupational Renal Diseases"

Professor of medicine, Professor of Preventive medicine and Community Health, UMDNJ- New Jersey Health care System, East Orange, New Jersey, Author of over two hundred papers including Poison in the Pot: The Legacy of Lead, Southern Illinois University Press. Carbondale, Ill., 1984, and Toxic Circles; Environmental Hazards from the Workplace into the Community, H.E. Sheehan and R.P. Wedeen, (eds), New Brunswick, Rutgers University Press, 1993.

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Li Xiao Phone: 0724-2337318
29 Xiangshan Avenue
Jinmen City 448000, Hubei

Title: "Leishuning (S311) Applied in the Treatment of Autoimmune Diseases"

Li Xiao, Male, aged 61, graduated from department of medicine, Zhongshan Medical University. Post-graduate in 1966 in the department of thoracic surgery, Zhongshan hospital. Now retired but employed as chief doctor in department of hematogenic immunity, No.2 People's Hospital, Jinmen City, Hubei. Mainly engaged in blood disease and autoimmune disease.

Thursday Afternoon Session

11:00 a.m. REGISTRATION

1:15 p.m. **WELCOME/MODERATOR: William J. Rea, M.D. & Charles T. Hinshaw, M.D.**

1:30 "Clinical Assessment of Taurine, a Modulator of Cellular Electrolytes", Jon Pangborn, Ph.D.

1:50 Q & A

2:00 "A Coming Catastrophe: Tobacco and Myocardial Infarction", Kaye Kilburn, M. D.

2:20 Q & A

2:30 "Mechanisms of How Current Cardiology Therapies Eventually Exacerbate Symptoms as Well as Create New Diseases", Sherry Rogers, M.D.

2:50 Q & A

3:00 BREAK WITH EXHIBITORS

MODERATOR: Bertie Griffiths, Ph.D.

3:30 "The Environmental Aspects of Cardiovascular Disease, Part I", William J. Rea, M.D.

3:50 Q & A

4:00 "Creating the Edible Landscape", Howard Garrett

4:20 Q & A

4:30 "Cardiac Parameters & Finger Temperature Response to Q-EEG-Neuro BioFeedback Rehabilitation of Brain-Injured Patients", Rima E. Laibow, M.D.

4:50 Q & A

5:00 **CASE STUDIES & PANEL/ MODERATOR: Allan Lieberman**

6:00 Adjourn

ABSTRACT INFORMATION & NOTES

Speakers Name: **Jon B. Pangborn, Ph.D.** Thursday, 1:30 p.m., June 8, 2000

SPEECH TITLE: **A Clinical Assessment of Taurine, a Modulator of Cellular Electrolytes**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To review physiological functions of taurine and to focus on its cardiovascular benefits. Following this, to instruct the audience on how to clinically assess taurine status.

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:**

A. Taurine has multiple, diverse physiological functions

B. In heart tissue, taurine helps to regulate electrolyte levels and arrhythmia can be due to insufficient taurine.

C. Low sulphur via hair analysis can be a prompt for assessing taurine via amino acid analysis.

D. Excessive urinary taurine can indicate wasting caused by beta-alanine in renal tubules.

4.) **References:**

- Welty, J.D. et al. "Effect of Taurine on Heart and Brain Electrolyte Imbalances" in Taurine: eds. R. Huxtable and A. Barbeau, Raven Press, 1976.
- Huxtable, R. "Metabolism and Function of Taurine in the Heart" in Taurine, ibid.
- Franconi F. et al. "Interaction between Organic Calcium-Channel Blockers and Taurine in Vitro and in Vivo", J. Pharm. Pharmacol.34 (1982) 329-330.
- Hayes K.C. et al."Taurine Modulates Platelet Aggregation in Cats and Humans", Am. J. Clin.Nutr. 49 (1989) 1211-16.
- Durlach J. and Y. Rayssiguier, "Donnees Nouvelles sur les Relations entre Magnesium et Hydrates de Carbone:, Magnesium 2 (1983) 174-91.
- Howard J.M.H. "Magnesium Deficiency in Peripheral Vascular Disease" J. Nut. Med. 1 (1990) 39-49.
- Wright C.E. et al. "Taurine: Biological Update" Ann. Rev. Biochem. 55 (1983) 427-53.
- Babior B.M. and C.A. Crowley. Chapt. 90 in The Metabolic Basis of Inherited Disease, 5th ed., McGraw-Hill (1983), 1962-1966.
- Scriver C.R. et al. Chapt. 27 in The Metabolic Basis of Inherited Disease, 4th ed., McGraw-Hill (1983), 1978) 534-535.
- Bremer H.J. et al. Disturbaces of Amino Acid Metabolism: Clinical Chemistry and Diagnosis, Urban & Schwarzenberg (1981) 168-169, 225.

Notes:

A CLINICAL ASSESSMENT OF TAURINE, A MODULATOR OF CELLULAR ELECTROLYTES

Jon B. Pangborn, Ph.D.

Discovered about 175 years ago, taurine (2-aminoethanesulfonic acid) was thought to be an inert artifact of human metabolism until about 1970 when its functions began to be understood. Abundant in human mother's milk but nearly absent in cow's milk, taurine was recognized as "semiessential" and even essential for children in the mid 1980s when it was added to commercial infant formula. In the 1990s, its antioxidant role as a scavenger of hypochlorite was investigated, and taurine was found to be a critical metabolite for phagocytosis. In the CNS, taurine helps to regulate the activity of two neurotransmitters, GABA and glutamic acid. In liver tissue, it combines with activated cholesterol ("cholyl-Coenzyme-A" to produce taurocholic acid. A primary bile acid that assists uptake of dietary lipids.

Not as well recognized is taurine's influence on cellular electrolyte levels and on body retention of magnesium. One of the earliest-discovered functions of taurine was that of regulating the flux of potassium, calcium and magnesium into cells while limiting cellular levels of sodium. In studies with rats, Welty et al. Discovered that heart muscle cells, when challenged with glucose, had normal electrolyte levels and normal function when taurine was adequate. When deficient in taurine, however, these same glucose-

stressed cells lost potassium and developed an abnormal Ca/Na ratio. Replenishment of taurine to the surrounding plasma corrected the potassium loss and rectified electrolyte ratios Ca/Na, Mg/Na, and K/Na.

The anti-arrhythmic action of taurine appears to be connected with its abundance in heart tissue in humans (and other mammals). In addition to beneficial actions for cardiac tissue, taurine is magnesium-sparing for the body globally. Normalized or healthy blood magnesium levels may decrease blood platelet aggregation and associated vascular disorders.

Assessment of taurine levels can be accomplished by amino acid analysis of fasting blood plasma or 24-hour urine. Fasting plasma represents the metabolic level (rather than a transient dietary influx), although it also is a point-in-time transport level. When low, inadequacy is certain. When normal in plasma, one should also check its primary metabolic precursor, cyst(e)ine. Low cysteine or cystine suggests limited taurine during periods of increased need. When high in plasma, one must check for blood cell homeolysis, leukocytolysis, infection, or toxicity that causes rupture of cell membranes. Urinary taurine must be interpreted with great care. Deficient urine taurine (with normal renal clearance), means deficiency. Elevated urine taurine can also mean deficiency due to wasting. Urinary taurine wasting typically occurs with elevated beta-alanine and possibly occurs with elevated beta-aminosobutyric acid. In renal tubules, beta-alanine blocks reabsorption of taurine. Elevated beta-alanine occurs with: (1) maldigestion and increased uptake of dietary peptides (anserine, carnosine), (2) infection or intestinal dysbiosis in which bacterial production is abnormally increase, and (3) catabolism of DNA and RNA as occurs with tissue necrosis or molignancy. Catabolism of tissue produces both beta-alanine and beta-aminoisobutyric acid.

Hair element analysis can suggest cystine and taurine insufficiency - the telltale result being deficient sulfur. Structurally, hair is about 14 to 15% cysteine and about 5% by weight sulfur. Low hair sulfur results often are found to correlate with methionine deficiency, cystinuria, or urinary taurine wasting, all of which are diagnosed by 24 hour urine amino acid analysis. Both methionine deficiency and urinary cysteine wasting imply limited reserves or capacity for taurine formation

The following clinical presentations are consistent with taurine insufficiency.

1. Signs of magnesium deficiency (muscle cramps, lower backache, constipation, fatigue, depression)

2. Hypersensitivity to chlorine, bleach or chlorinated water

3. Cardiac arrhythmia

4. Steatorrhea

5. Elevated cholesterol

6. Abnormally enhanced inflammation during infections

7. Frequent infections, leukocytolysis or leukopenia

8. Maldigestion

9. Seizures

ABSTRACT INFORMATION & NOTES

Speakers Name: **Kaye H. Kilburn, M.D.** Thursday, 2:00 p.m., June 8, 2000

SPEECH TITLE: **A Coming Catastropy: Tobacco and Myocardial Infarction**

The information below has been provided by the speaker.

1.) **Goals and objectives:** Why is a new strategy needed to reduce deaths from heart disease worldwide.

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:** That coronary heat disease is the major killer of mankind at the dawn of Y2K. Its spread into developing countries coincided with that of tobacco smoking and automobile exhaust. Prevention is the only answer. The US must stop growing tobacco and exporting cigarettes and their manufacturing technology.

4.) **References:**

1. Horton R: Future of European cardiology. Lancet 1999;354:791-792.
2. Fuster V: Epidemic of cardiovascular disease and stroke: the three main challenges. Circulation 1999;99:1132-1137.
3. Rosamond WD, et al.: Trends in incidence of myocardial infarction and mortality due to coronary artery disease 1987-1994. New Eng J Med 1998;339:380-385.
4. Henneken CH: Increasing burden of cardiovascular disease. Circulation 1998;97:1095-1102.
5. Reddy KS and Yusuf S: Emerging epidemic of cardiovascular disease in developing countries. Lancet 1998;351:586-590.

Notes:

A COMING CATASTROPY: TOBACCO AND MYOCARDIAL INFARCTION

Kaye H. Kilburn, M.D.

University of Southern California, Keck School of Medicine

CHD-AMI is world's most important disease, killing 6,300,000 people yearly, 3.5 million of these in developing countries (1,2). It was so rare in the previous century that it was considered an exotic variety of heart disease that stimulated speculation but no action. Concern emerged in the first quarter of this century as more patients were diagnosed, it blossomed and caught-on. The CHD-AMI plague emerged from obscurity to become epidemic by the 1950's (3). It was recognized first in America and in Britain, crept to Europe and engulfed the world. From rare in 1990 the diagnosis became common by 1950. Its upward trend coincided with those for cigarette smoking and burning hydrocarbon fuels in automobiles. The causes of CHD-AMI appear elusive despite 50 years of intensive research. Perhaps we need to think of it as a chemical contagion as the abrupt emergence of CHD-AMI in this century is poorly explained by its agreed upon risk factors.

The evidence that tobacco smoke causes CHD-AMI developed by 1958 and was strengthened when the deaths decreased in men and then women who quit smoking. Evidence of parallel effect from air pollution from fossil fuel burning is deduced. Worldwide bankruptcy and chaos from billions of deaths and massive numbers disabled can be avoided only by prevention of smoking. A strategy is needed to stop cigarette production and tobacco growing. To do this simple job may take broader social action like that leading to the exclusion of asbestos from commerce. It must be done as time is running out.

Slide 1 Coronary

A Coming Catastrophy: tobacco and myocardial infarction

Kaye H. Kilburn, MD., Professor of Medicine
University of Southern California, Keck School of Medicine,
2025 Zonal Avenue, CSC 201, Los Angeles, CA 90033 and Neuro-Test, Inc.

Slide 2 **A Little History**

- 1842 Marshall Hall - Lecture, Coronary Circulation - Sudden death
- 1842 John Ericksen - exper. In dogs and rabbits ligature of coronary arteries causes deaths
- 1887 Cohnheim - timed 30 - 40 sec. pulse intermittent ventricles stopped in 105 sec.
- Cruveilheir 1842, Rokitansky 1856 Virchow described myocardial infarcts, MI
- 1863 Boettger - 62 cases of heart rupture

- 1876 Hammer - heart block 40, 23, 20, 16 due to CT throm. Obst. Sinus Valsalva - autopsy
- 1882 Huber - 17 histories of MI - plugs
- 1910 Osler - 17 fatal angina + autopsy
- 1910 Obrastzow, Streschesko - 3 CT-kiev
- 1911 Hochhaur - 4 CT-Cologne

Slide 3

- 1912 James Herrick - diagnosed CT-MI during life
- 1919 Herrick - 4 more in 200 angina pts.
- 1927 Wearn - 10 Boston pts.
- 1928 Bedford - Britain 100 pts.
- 1929 Levine - Boston145 pts.
- 1939 George Dock - freq. Increasing
- 1942 Herrick - nothing by auscultation etc.
- 1948 Yater - 450 ages 18-39 AFIP-VA
- 1954 Hammond-Horn - cig. smoke
- 1985-91 Fye - mystery of delayed dx.
- 2000 - Kilburn - hypothesis

Slide 4 Prior to World War II, coronary artery disease in men under 30 years age was regarded as rare, and in men between 30 & 40 years of age, as uncommon. Yater, 1948 AHJ

Slide 5 **Lifetime Risk.....**

Lifetime risk at age 40 years it is one in three for women. Even at age 70 years it is one in three for men and one in four for women.

Measurement

- MacKenzie - polygraph
- Roentgen - x-ray
- Einthoven, Lewis - ECG
- Riva-Rocci, von Rechlinghaus - BP cuff 4.5 v 12.7
- Forssmann, Cournand and Richards - catheter

Slide 6 **Deaths worldwide**

- 6,300,000 CT-MI
- 4,400,000 Stroke
- 66% of diabetic deaths CT

Costs

- USA - 1 million bypass operations/year
- $51.2 billion cost, $15 billion for surgery

Fact or Fallacy

- Comparisons of rates
- Second hand smoke
- Trends in air pollution
- Air pollution 1958
- 1980 lung cancer rate in NS exceeded the rate in smokers in 1940-50

Slide 7 **Factors**

- Cigarette smoking - men & MD's
- Hypertension - Kenya
- Elevated cholesterol in ½ of CT

Air pollution and other inhaled chemicals

Chemical Use in USA

- 1950 - 2 billion pounds
- 1990 - 300 billion pounds

Oil Use

- 1999 - 3 billions tons, 6 trillion pounds
- Automobiles
- 26 million 1945
- 130 million 1990

Slide 8 **Toxic Oil Syndrome**

- Spain 1981, 25,000 people
- Respiratory distress syndrome

- Rapeseed oil and aniline
- Pulmonary hypertension in 20%
- Cor pulmonale & endothelial damage 40
- Coronaries 11, conducting system, sinus node (8 resemble lupus, 4 scleroderma)
- degenerative lesions

Unifying Concept: INFLAMMATION

- Endothelial injury, small blood vessel loss
- Inhaled chemicals stimulated lung cells, neutrophils, endothelial cells, macrophages
- Cells release pressor substances and mediators that initiate and sustain inflammation

Results

- Hypertension
- Plaques
- Thrombosis

Slide 9 **Coronary Plaques**

1. Endothelial permeability mediated by NO, prostacyclin, PDGF...

2. Fatty streak formation lipid laden macrophages

3. Advanced lesion - plaque fibrous cap over necrotic core

4. Unstable fibrous plaque

    hemorrhage, proteolysis

    thrombosis, platelets plus clotting cascade

Realities

1. Cold turkey withdrawal form cigarette smoking works for individuals.

2. Education is too slow and does not work well for youth.

3. Tobacco is the worlds most dangerous product.

4. Profit is a poor justification for immoral corporate decisions.

Slide 10 **Solutions**

- Cease manufacturing cigarettes.
- Reduce fossil fuel burning, H2 fuel
- Cut chemical use.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Sherry S. Rogers, M.D.** Thursday, 2:30 p.m., June 8, 2000

SPEECH TITLE: **Mechanisms of How Current Cardiology Therapies Eventually Exacerbate Symptoms as Well as Create New Diseases**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To expose the physician to the little-known mechanisms of biochemical destruction of commonly prescribed core treatment cardiac drugs.

2.) **Outline of talk/abstract:** As a brief example, calcium channel blockers increase heart attach and cancer rate as well as cause deterioration of the white matter of the brain, while HMG-CoA inhibitors turn off the body's path to generate coenzyme Q10, thereby leading the patient into heart failure.

3.) **Conclusion of what is to be learned:** That cardiac drugs actually generate worsening of the underlying condition as well as promote new symptoms.

4.) **References:**

1. Newman TB, Hulley SB, Carcinogenicity of lipid-lowering drugs, JAMA, 1996;275;1:55-60
2. Folkers K, Longsjoen P, Willis RA, et al, Lovastatin decreases coenzyme Q10 levels in humans, Proc Natl Acad Sci 1990;87:8931-4
3. Fitzpatrick AL, Daling JR, Weissfeld JL, et al, Use of calciu-channel blockers and breast carcinoma risk in postmenopausal women, Cancer 1997,80:1438-47
4. Psaty BM, Heckbert SR, Furberg CD, et al, The risk of myocardial infarction associated with anti-hypertensive drug therapes, JAMA 1995;274:620-25

Notes:

MECHANISM OF HOW CURRENT CARDIOLOGY THERAPIES EVENTUALLY EXACERBATE SYMPTOMS AS WELL AS CREATE NEW DISEASES

Sherry A. Rogers, M.D.

ABSTRACT INFORMATION & NOTES

Speakers Name: **William Rea, MD** Thursday, 3:30 p.m., June 8, 2000

SPEECH TITLE: **The Environmental Aspects of Cardiovascular Disease, Part I**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To better understand the environmental aspects of cardiovascular disease.

2.) **Outline of talk/abstract:** To learn how to use in the office the knowledge acquired to enhance the patient's treatment.

3.) **Conclusion of what is to be learned:** The outcome of these problems is very successful when causes are found and eliminated.

4.) **References:**

Notes:

THE ENVIRONMENTAL ASPECTS OF CARDIOVASCULAR DISEASE, PART I

William J. Rea, MD, FACS, FAAEM
Environmental Health Center - Dallas

There is a broad spectrum of patients whose cardiovascular system is affected by environmental pollutants. These pollutants seem to fall in the general category of organochlorine and organophosphate pesticides, solvents (especially the chlorinated ones), phenols and formaldehydes. Our series of patients with cardiovascular disease triggered by pollutants is presented. A spectrum of cardiac arrhythmias, heart failure, dissecting aneurysm and cardiomyopathy is reviewed. Nutritional as well as environmental causes will be discussed.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Howard Garrett** Thursday, 4:00 p.m., June 8, 2000

SPEECH TITLE: **Creating the Edible Landscape**

The information below has been provided by the speaker.

1.) **Goals and objectives:** Teaching how to plant the landscape to consist totally of medicinal and culinary plants.

2.) **Outline of talk/abstract:** Design bed preparation, trees, shrubs, ground covers, vines, perennials, annuals.

3.) **Conclusion of what is to be learned:** The entire landscape can be made of useful plants and grown with organic techniques

4.) **References:**

Dirt Doctor's Dirt newsletter, Texas Organic Vegetable Gardening Book, Texas Bug Book, The Organic Manual

Notes:

CREATING THE EDIBLE LANDSCAPE

Howard Garrett

(These are all herbs - not 'erbs)

SHADE TREES:

Ginkgo - tea from leaves
Jujube – fruit
Linden - tea from flowers
Mulberry – fruit
Pecan - edible nuts
Persimmon – fruit
Walnut - edible nuts

SHRUBS:

Agarita - fruit for wine
Althea - edible flowers
Bay - tea and, food seasoning from leaves
Germander - freshens air indoors
Pomegranate - edible fruit
Turk's cap - flowers and fruit for tea

ANNUALS:

Begonias - edible flowers
Daylilies - edible flowers
Dianthus - edible flowers

Ginger - food, seasoning and tea from roots
Hibiscus - edible flowers
Johnny jump-ups - edible flowers
Nasturtium - edible leaves
Pansies - edible flowers
Peanuts - edible nuts
Purslane - edible leaves
Sunflower - edible seeds and flower petals

VINES:

Beans and Peas - edible pods and seed
Gourds - dippers and bird houses
Grapes - food (fruit and leaves)
Luffa - sponges from the fruit, edible flowers
Malabar spinach - edible foliage
Passion flower - edible fruit, tea from leaves

ORNAMENTAL TREES:

Apple - fruit and edible flower petals
Apricot - fruit and edible flower petals
Citrus - edible fruit
Crabapple - fruit and edible flower petals
Fig - fruit and edible flower petals
Mexican plum – fruit
Peach - fruit and edible flower petals
Pear - fruit and edible flower petals
Persimmon – fruit
Plum - fruit and edible flower petals
Redbud - edible flowers
Rusty blackhaw viburnum - edible berries
Witchhazel - tea from leaves

PERENNIALS:

Anise hyssop- edible flowers, foliage for tea
Blackberries - edible berries, foliage for tea
Chives - edible foliage and flowers
Garlic - edible flowers, greens and cloves
Hibiscus - edible flowers
Hoja santa - leaves for cooking with meats
Horsemint - insect repellent
Jerusalem artichoke - roots for food
Lavender - teas and insect repellent
Monarda - edible flowers and leaves for teas

Peppers - edible fruit
Purple coneflower - all plant parts for teas
Rosemary - food and tea from leaves and flowers
Roses - petals and hips for tea
Salvia - edible flowers, foliage for teas
Sweet marigold - food, flavoring and tea from leaves and flowers
Tansy - chopped and crushed foliage repels ants
Turk's cap - flowers & fruit for tea

GROUND COVERS:

Clover - tea from leaves and flowers
Creeping thyme - teas and food flavoring
Gotu kola - tea from leaves
Mints - food and teas from flowers and leaves
Oregano - teas and food flavoring
Violets - leaves in salads and tea from flowers and leaves **Note:** Pregnant women should avoid all strong herbs and no plant should be ingested in excess by anyone. None of these should be eaten unless they are being grown organically.

EDIBLE FLOWERS

Aloe vera, althea, apple blossoms, arugula, basil, begonia, borage, broccoli, calendula, chicory, chives - onion and garlic, clover, coriander, dandelion, dill, elderberry, English daisy, fennel, hyssop lavender, lemon, lilac, mint, monarda - red flowered *M. didyma*, mum (base of petal is bitter), mustard, okra, orange, oregano, pea (except for sweet peas), pineapple sage, radish, redbud, rosemary, scented geranium, society garlic, sweet woodruff, squash blossoms, thyme, violet, winter savory, yucca (petals only)

RULES FOR EDIBLE FLOWERS

1.) Not all flowers are edible. Some are poisonous. Learn the difference.

2.) Eat flowers only when you are positive they are edible and non-toxic.

3.) Eat only flowers that have been grown organically, toxic materials collect in the reproductive plant parts.

4.) Do not eat flowers from florists, nurseries or garden centers unless you know they've been maintained organically.

5.) Do not eat flowers if you have hay fever, asthma or allergies.

6.) Do not eat flowers growing on the side of the road.

7.) Remove pistils and stamens from flowers before eating. Eat only the petals, especially of large flowers.

8.) Introduce flowers into your diet the way you would new foods to a baby - one at a time in small quantities.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Rima E. Laibow, MD** Thursday, 4:30 p.m., June 8, 2000

SPEECH TITLE: **Cardiac parameters and finger temperature response to Q-EEG-Neuro BioFeedback rehabilitation of brain-injured patients**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To inform participants of the potential cardiac rehabilitation through Neuro Bio Feedback

2.) **Outline of talk/abstract:** Definition of the problem, clinical study design and population, introduction to treatment methodology discussion of results and implications fro treatment and research.

3.) **Conclusion of what is to be learned:** Cardiac Parameters can be normalized through the use of nontoxic, non invasive, pharmaceutical-free treatment. This treatment effect appears to be both durable and robust.

4.) **References:**

A.) Gillespie, C.R., Peck, F.F., 1980. The effect of biofeedback and guided imagery on finger temperature. Biol. Psychol., 11(3-4), 235-247.

B.) Ikemi, A., Tomita, S., Hayashida, Y., 1988. A thermographic analysis of the warmth of the hands during the practice of self-regulation method. Psychother. Psychosom., 49-52.

C.) Laibow, R.E., Bounias, M., Stubblebine, A.N., Sandground, H., 1996. Rehabilitation of brain injured adults and adolescents through neural therapy (voluntary regulation of EEG activity. In: Effective strategies fro Assessment and Intervention, Proc. 20th Annu. Postgraduate Course on Rehabilitation of the Brain Injured Adult and Child, Office of Continuing Medical Education, Virginia Commonwealth University, Medical College, Williamsburg, June 6-9, 1996, 153-155.

D.) Bounias, M. Laibow, R.E., Stubblebine, A.N., Sandground, H., and Bonaly, A. 1997a. The duration of NeuroBioFeedback treatment as a function of both the initial load of clinical symptoms and the rate of rehabilitation of brain injured patients. Submitted.

E.) Laibow, R.E., Stubblebine, A.N., Sandground, H., Bonaly, A. and Bounias, M., 1997a. Changes in EEG parameters following Q-EEG-NeuroBioFeedback treatment of brain injured patients. Submitted.

F.) Laibow, R.E., Bounias, M., 1997b. Neurobiofeedback. In: The textbook of Complementary and Alternative medicine, W.B. Jonas and J.S. Levin Eds., Williams & Wilkins, Baltimore, 22 pp. In press.

G.) Lubar, J.F., Deering, W.M., 1981. Behavioral approaches to neurology. Academic Press, New York.

H.) Luthe W, 1969, Autogenic therapy. Vol. 4 New York: Grune and Stratton, 1969.

Mandelzys, N., Lane, E.B., and Marceau, R., 1981. The relationships of violence to alpha levels in a biofeedback training paradigm. J. Clin. Psychol., 37, 202-209.

G.) McGrady, A.V., Yonker, R., Tan, S.Y., Fine, T.H., Woerner, M., 1981. The effect of biofeedback-assisted relaxation training on blood pressure and selected biochemical parameters in patients with essential hypertension. Biofeedback Self Reful, 6(3), 343-353.

H.) Mulholland, T., 1995. Human EEG, behavioral stillness and biofeedback. International Journal of Psychophysiology, 19, 263-279.

I.) Nuwer, M.R. 1988. Quantitative EEG: II. Frequency analysis and topographic mapping in clinical settings. J. Clin. Neurophysiol., 5(1), 45-85.

J.) Okouchi, H., Sugiwaka, H., 1995. Transitory decreases in skin temperature during temperature increase training: possible explanation, Shinrigaku Kenkyu, 66(1), 48-51.

K.) Remond, A. and Remond, A., 1994. Biofeedback. Principes et applications. Masson, Paris, 35-68.

L.) Sappington, J.T., Fiorito, E.M., 1995. Thermal feedback in Raynaud's phenomenon secondary to systemic lupus erythematosus: long term remission target symptoms. Biofeedback Self Regul, 10(4), 335-341.

M.) Schwartz G., Disregulation and systems theory: a behavioral framework for biofeedback and behavioral medicine. In: Birbaumer N, Kimmel HD eds. Biofeedback and Self-Regulation. Hillsdale, J.J.: Lawrence Erlbaum Associates, 1979: 19-48.

N.) Shapiro, D., 1979. Biofeedback and behavioral medicine: an overview. Psychother. Psychosom., 31(1-4),24-32.

CARDIAC PARAMETERS AND FINGER TEMPERATURE RESPONSE TO Q-EEG-NEUROBIOFEEDBACK REHABILITATION OF BRAIN-INJURED PATIENTS

Rima E. Laibow, MD

Medical Director, **Alexandria Institute of Natural and Integrative Medicine, Croton on Hudson, NY**

A population of 27 brain injured patients were treated by computer-assisted quantitative elecroencephalographic Neuro BioFeedback (Q-EEG-NBF). In parallel to targeted changes in EEG power spectra, secondary effects were observed for heart rate as well as for systolic and to a lesser extent for diastolic pressures, generally in the sense of up- and down-regulation of extreme (resp. lower and higher than average) values. Finger temperature, reflecting an improvement of blood circulation, proved to increase both in rate and amplitude from the beginning to the end of treatments, correlatively with the rate of patient's rehabilitation. NBF therefore achieves beneficial side-effects in addition to our complement of the initially targeted improvements.

Key words. Systolic and diastolic pressure and ratios; heart rate; finger temperature; clinical classes.

Running Title. Cardiovascular side-response to Q-EEG-NBF.

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Friday Session

8:15 **ANNOUNCEMENTS/MODERATOR: Richard Jaeckle, M.D.**
8:30 "Global Environmental Update 2000", Doug Seba, Ph.D.
8:50 Q & A
9:00 "Environmental Signals and Immune Response", Kou Sakabe, M.D.
9:20 Q & A

9:30 "Growing Ornamentals and Food Crops the Natural Way", Howard Garrett
9:50 Q & A

10:00 BREAK WITH EXHIBITORS

MODERATOR: Kay Kilburn, M. D.
10:30 "The Diagnosis and Therapy of EM Hypersensitivity", Cyril Smith, Ph.D.
10:50 Q & A
11:00 "Lead and Hypertension: Who Cares?", Richard Wedeen, M.D.
11:20 Q & A
11:30 "Effectiveness of Estrogen replacement and Intravenous Magnesium Therapy to Correct Dyshomeostasis and Dysfunctioning Vascular Endothelium", Kalpanna Patel, M.D.
11:50 Q & A
12:00n BUFFET LUNCH WITH THE EXHIBITORS

MODERATOR: Charles Hinshaw, M.D.
1:30 "Heart Rate Variability in the Food and Chemically Sensitive Patient", Jean Monro, M. D.
1:50 Q & A
2:00 "Antioxidant Adaptation, Cancer and Cardiovascular Disease", Stephen Levine, Ph.D.
2:20 Q & A
2:30 "The Environmental Medicine Approach to Solving Recalcitrant Cardiology Conditions", Sherry Rogers, M.D.
2:50 Q & A

3:00 BREAK WITH EXHIBITORS

3:30 "Diagnosis and Treatment of Cell-Invasive Bacterial Infections in CFS, FMS, Gulf War Illness and Rheumatoid Arthritis", Prof. Garth L. Nicolson
3:50 Q & A
4:00 "The Use of the Halstead-Reitan Neuropsychological Test Battery to Measure Neurotoxic Effects in Chemical -Exposed Individuals", Nancy Didriksen, Ph.D.
4:20 Q & A
4:30 CASE STUDIES & PANEL/ MODERATOR: William J. Rea, M.D.
Case # 1 Tonya Callaway
Case # 2 Tom Croley, Ph.D.
Case # 3 Richard Jaeckle, M.D.
6:00 Adjourn

ABSTRACT INFORMATION & NOTES

Speakers Name: **Doug Seba, Ph.D.** Friday, 8:30 a.m., June 9, 2000

SPEECH TITLE: **Global Environmental Update 2000**

The information below has been provided by the speaker.

1.) **Goals and objectives:**

- To review some of the major environmental pollutants of the last 1000 years.
- To review the two major environmental phenomenon at the close of this millennium: Global warming and increasing dust.
- To review health implications of these two events.

2.) **Outline of talk/abstract:** Physical, Chemical and Biological events of note are covered with focus on fate and transport mechanisms that re-concentrated chemicals.

3.) **Conclusion of what is to be learned:** That as the world warms, increasing dust loads and their components of minerals, nutrients, fungal spores and other pathogens, can have a plethora of adverse environmental and health effects at vast distances from their origin.

4.) **References:**

- "Pesticides in the Lower Atmosphere of the Northern Equatorial Atlantic Ocean." by Douglas B. Seba and J. M. Prospero. Atmospheric Environment 5:1043-1050, 1971.
- "Surface Slicks as Concentrators of Pesticides in the Marine Environment" by Douglas B. Seba and E. F. Corcoran. Pesticides Monitoring Journal 3:190-190, 1969.

Notes:

GLOBAL ENVIRONMENTAL UPDATE 2000

Douglas B. Seba

Independent Marine Scientist, Key West, FL

Up until the 13th or 14th century, people could see the planet Venus during the day the same way we can still see the moon during daylight. Since that time, global pollution, mainly from atmospheric dust loadings, has prevented that. Thus, for over 25 generations back from present, no one in the whole world, has been able to draw one breath of air as pure as our ancestors did throughout their lives for the last 25,000 years. Thus, dust could arguable be said to be the biggest pollutant of this millennium. Certainly radionuclides and synthetic chemicals should be given runner-up status but they have come on the scene only in the last 10% of this time period.

Also, as we exit this millennium we find a global warming trend that probably has not existed for several millennia. The trend is real, only the cause, and therefore what action to take, is in confusion. The apparent only reason the earth is not already much warmer is that the missing heat is being stored in oceans, a temporary phenomenon.

Global warming has diverse ecosystem effects and related health issues which will be explored. In particular, increasing dust loads from Africa to the eastern US will be explored as a contemporary example. Dust loadings in the last year have been the greatest since systematic measurements were begun over four decades ago. This has resulted in several new Internet sites being established. The best education site which is a discussion forum and catalog of web resources on atmospheric dust is:

http: //capita.wust1.edu/Databases/UserDomains/SaharaDust2000/

and the best government site to see satellite imagery is:

http: // seawifs.gsfc.nasa.gov/SEAWIFS/HTML/dust.html

Human health effects from this African dust is being coordinated by Dr. Eugene Shinn, Center for Coastal Geology, U.S. Geological Survey, 600 4th Street South St. Petersburg, FL 33701. E-mail: gene@wayback.er.usgs.gov Telephone: 727/803-8747 Extension 3030.

The author wishes to thank the Academy of Marine Sciences in Ft. Lauderdale for its continuing support of this research.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Kou Sakabe, MD** Friday, 9:00 a.m., June 9, 2000

SPEECH TITLE: **Environmental Signals of Immune Response**

The information below has been provided by the speaker.

1.) **Goals and objectives:** Elucidation of immune disrupting mechanism of environmental estrogens (i.e., environmental signals) and related compounds for health risk assessment.

2.) **Outline of talk/abstract:** a) strong inhibitory effect of environmental signals on the C kinase activity of mitogen-stimulated lymphocytes was observed; b) environmental signals studied all significantly reduced p34cdc2 kinase activities; c) environmental signals inhibit development in the both T- and B-lymphocyte stem cell compartments.

3.) **Conclusion of what is to be learned:** The cytoplasmic signal-generating system in developing or mitogen-treated lymphocytes are inhibited by environmental signals, and the defect occurs at all stages int eh sequence of events leading to DNA synthesis, cell proliferation and cell differentiation.

4.) **References:**

1. Int.J.Immunopharmacol. Vol. 20(4-5),205-212,1998.
2. Int.J.Immunopharmacol. Vol.21(12),861-868,1999.

3. Pathophysiol. Vol.6(1),231-236,2000.

Notes:

ENVIRONMENTAL SIGNALS AND IMMUNE RESPONSE

Kou Sakabe, MD, Mikio Miyata, Satoshi Ishikawa, MD

Environmental Medical Center, Kitasato Institute Hospital, Tokyo, Japan

Substantial evidence has been accumulated to support the sex hormonal regulation of immune functions. They are mainly based on the following observations: I) the existence of sexual dimorphism in immune response, ii) alteration of immune response by sex steroid replacement, iii) alteration of immune response during pregnancy, and iv) existence of sex steroid receptors in the immune organs which affect T or B lymphocyte differentiation and function. This evidence strongly supports the hypothesis that sex steroid such as estrogen, plays a strong role in the immune functions of many animals and humans. Although this family of natural estrogens are steroidal in structure, a variety of exogenous non-steroids has been found to act like estrogens. In fact, estrogenic zenobiotics (i.e., environmental estrogens), which are one of the endocrine disruptors, have been implicated in a number of human health disorders. These hormonally active agents (HAAs) are derived from a number of relatively common and abundant sources such as pesticides, insecticides, plastics, combustion by products, plants and agricultural products. However, little is known about the pharmacological or toxicological effects on immuno-competent cells of exposure to these HAAs especially in reference to the cause of animal and human immune disorders. To address this issue, the present study focuses on the effect of HAAs on differentiation and function of animal and human lymphocytes. The result obtained are as follows: a)the proliferation of peripheral blood lymphocytes (PBL) in response to IL-2 was mediated by C kinase activity, but a strong inhibitory effect of HAAs on the C kinase activity of IL-2-stimulated PBL was observed; b) cytoplasmic extracts of IL-2-stimulated PBL showed high activity as an activator of DNA replication as well as increased levels of the mitosis stimulator p34cdc2 kinase, and the HAAs studied all significantly reduced these activities; c) HAAs inhibit development in the both T-and B-lymphocyte stem cell compartments (there is evidence for apoptosis in HAAs inhibition of immature lymphocyte development). The results suggest that the cytoplasmic signal-generating system in developing or mitogen-treated lymphocytes are inhibited by HAAs, and that the defect occurs at all stages in the sequence of events leading to DNA synthesis, cell proliferation and cell differentiation.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Howard Garrett** Friday, 9:30 a.m., June 9, 2000

SPEECH TITLE: **Growing Ornamentals and Food Crops the Natural Way**

The information below has been provided by the speaker.

1.) **Goals and objectives:** Converting the world to organic - Eliminating the use of toxic chemical fertilizers and pesticides.

2.) **Outline of talk/abstract:** History, basic organic program, case studies, research using organic amendments, organic pest control

3.) **Conclusion of what is to be learned:** Organic programs are less toxic, more effective, cost effective and the only way to improve the heatlh of environment

4.) **References:**

The Organic Manual, Texas Organic Vegetable Gardening, Texas Bug Book

Notes:

GROWING ORNAMENTALS AND FOOD CROPS THE NATURAL WAY

Howard Garrett

SOIL TESTING - Send soil samples to Texas Plant and Soil Lab in Edinburg, TX, 956-383-0739 for organic recommendations. Another way to test the soil is to dig a cubic foot of soil and sift it back into the hole. If you don't see about 10 earthworms, you need to do more of what's listed below.

PLANTING - Prepare new planting beds by scraping away existing grass and weeds, adding a 4-6" layer of compost, lava sand at 40-80 lbs., organic fertilizer at 20 lbs. horticultural cornmeal 10-20 lbs./1,000 sq. ft. and tilling to a depth of 3" into the native soil. Excavation and additional ingredients such as concrete sand, topsoil and pine bark are unnecessary and can cause problems. More compost is needed for shrubs and flowers than for groundcover. Add Texas greensand to black and white soils and high-calcium lime to acid soils. Soft rock phosphate is an effective amendment for all soils.

FERTILIZING - Apply an organic fertilizer 2-3 times per year. During the growing season, spray turf, trees and shrub foliage, trunks, limbs and soil at least monthly with Garrett Juice. Add lava sand annually at 40-80 lbs./1,000 sq. ft.

MULCHING - Mulch all shrubs, trees and ground cover with 3-5" of, shredded tree trimmings or shredded hardwood bark to protect the soil, inhibit weed germination, decrease watering needs and mediate soil temperature. Mulch vegetable gardens with 8" of alfalfa hay, rough-textured compost or shredded native tree trimmings. Avoid Bermuda hay because of the possibility of broadleaf herbicide contamination. Shredded native cedar is the best of all mulches.

WATERING - Adjust schedule seasonally to allow for deep, infrequent wafering in order to maintain an even moisture level. Start by applying about 1" of water per week in the summer and adjust from there. Water needs will vary from site to site and from

season to season. Add 1 tablespoon natural vinegar per gallon when watering pots, unless water is acid.

MOWING - Mow weekly, leaving the clippings on the lawn to return nutrients and organic matter to the soil. General mowing height should be 2-1/2" or taller. Put occasional excess clippings in compost pile. **Do not ever bag clippings**. **Do not let clippings ever leave the site.** Mulching mowers are best if the budget allows. Do not use line trimmers around trees.

WEEDING - Hand pull large weeds and work on soil health for overall control. Mulch all bare soil in beds. AVOID SYNTHETIC HERBICIDES, especially pre-emergents, broad-leaf treatments and soil sterilants. These are unnecessary toxic pollutants. Spray broadleaf weeds as a last resort with full strength vinegar, and citrus mix or remove mechanically. Commercial organic herbicides are now on the market.

PRUNING - Remove dead, diseased and conflicting limbs. Do not over prune. Do not make flush cuts. Leave the branch collars intact. Do not paint cuts except on red oaks and live oaks in oak-wilt areas when spring pruning can't be avoided. Remember that pruning cuts hurt trees. Pruning is done for your benefit, not for the benefit of the trees.

COMPOST MAKING - Compost, Nature's own living fertilizer, can be made at home or purchased ready-to-use. A compost pile can be started any time of the year in sun or shade. Anything once living can go in the compost - grass clippings, tree trimmings, food scraps, bark, sawdust, rice hulls, weeds, nut hulls and animal manure. Mix the ingredients together and simply pile the material on the ground. The best mixture is 80% vegetative matter and 20% animal waste, although any mix will compost. Since oxygen is a critical component, the ingredients should be a mix of coarse and fine-textured material to promote air circulation through the pile. Turn the pile once a month if possible, more often speeds up the process but releases nitrogen to the air. Another critical component is water. A compost pile should be roughly the moisture of a squeezed-out sponge to help the living organisms thrive and work their magic. Compost is ready to use as a soil amendment when the ingredients are no longer identifiable. The color will be dark brown, the texture soft and crumbly and it will smell like the forest floor. Rough, unfinished compost can be used as a top-dressing mulch around all paintings.

MANURE COMPOST TEA - Manure compost tea is an effective foliar spray because of many mineral nutrients and naturally occurring microorganisms. Fill any container half full of compost and finish filling with water. Let the mix sit for 10-14 days and then dilute and spray on the foliage of any and all plants. How to dilute the dark compost tea before using depends on the compost used. A rule of thumb is to dilute the leachate down to one part compost liquid to 4 to 10 parts water. The ready-to-use spray should look like iced tea. Be sure to strain the solids out with old pantyhose, cheese cloth or floating row cover material. Full strength tea makes an excellent fire ant mound drench when mixed with 2 oz. molasses and 2 oz. citrus oil per gallon. Add vinegar, molasses and seaweed to make Garrett Juice.

CONTROLLING INSECTS - Aphids, spider mites, whiteflies & lacebugs: release ladybugs and green lacewings regularly until natural populations exist. Garrett Juice

and/or garlic-pepper tea (recipes below) are effective controls. Use strong water blasts for heavy infestations. **Caterpillars and bagworms:** release trichogramma wasps. Spray *Bacillus thuringiensis* (Bt) as a last resort. **Fire ants:** Drench mounds with Garrett Juice plus citrus oil and release beneficial nematodes. **Grasshoppers:** Eliminate bare soil, apply beneficial nematodes, and then dust or spray one or more of the following: self-rising flour, natural diatomaceous earth, fire ant control formula. Encourage biodiversity and feed the birds. **Grubworms:** beneficial nematodes and general soil health is the primary control. **Mosquitoes:** *Bacillus thuringiensis* 'Israelensis' for larvae in standing water. Spray citrus oil or garlic-pepper tea for adults. Lavender, vanilla, citronella and eucalyptus also repel mosquito adults. **Slugs, snails, fleas, ticks, chinch bugs, roaches, crickets:** spray or dust diatomaceous earth products and crushed red pepper. Citrus oil also kills these pests. For more details on pest control, check out the new *Texas Bug Book*.

CONTROLLING DISEASES - Black spot, brown patch, powdery mildew and other fungal problems: best control is prevention through soil improvement, avoidance of high-nitrogen fertilizers and proper watering. Spray Garrett Juice plus garlic and/or neem. Baking soda or potassium bicarbonate can also be added. Treat soil with horticultural cornmeal at about 20 lbs./1,000 sq. ft. Alfalfa meal and mixes containing alfalfa are also good disease fighters.

GARLIC-PEPPER TEA INSECT REPELLENT - In a blender with water, liquefy 2 bulbs of garlic and 2 cayenne or habanero peppers. Strain away the solids. Pour the garlic-pepper juice into a 1 gallon container. Fill the remaining volume with water to make one gallon of concentrate. Shake well before using and add 1/4 cup of the concentrate to each gallon of water in the sprayer. To make garlic tea, simply omit the pepper and add another bulb of garlic. For additional power add 1 tablespoon of seaweed and molasses to each gallon. Always use plastic containers with loose fitting lids for storage.

GARRETT JUICE (foliar spray and soil drench) - Mix the following per gallon of water: 1 cup of compost tea or liquid humate, 1 ounce liquid seaweed, 1 ounce blackstrap molasses, 1 ounce apple cider vinegar. **To make a mild insect control product, add 1 oz. of citrus oil per gallon of spray. To make the fire ant killer, add 2 oz. of citrus oil per gallon.** When spraying the foliage of plants, don't use over 2 oz. of citrus oil per gallon of spray. This mixture also works as a soil detox.

DIRT DOCTOR'S POTTING SOIL - 5 parts compost, 4 parts lava sand, 3 parts peat moss, 2 parts cedar flakes, 1 part soft rock phosphate, 1 part earthworm castings, ½ wheat bran/cornmeal soil amendment, ¼ part organic fertilizer, ¼ part sul-po-mag, ¼ part Texas greensand. This is a very powerful potting soil and needs no additional fertilizer. It is also too strong to use for most interior house plants.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Cyril W. Smith Ph.D.** Friday, 10:30 a.m., June 9, 2000

SPEECH TITLE: **The Diagnosis and Therapy of EM Hypersensitivity**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To provide insight into the clinical aspects of electromagnetic hypersensitivity.

2.) **Outline of talk/abstract:** Frequencies are presented as the "triggers" of reactions (including heart sensitivities) to the EM environment. The symptoms, their diagnosis and treatments available, patient support needs, and synergism between the chemical and electromagnetic aspects of the reactions are to be discussed.

3.) **Conclusion of what is to be learned:** How to recognise EM triggered reactions in your patients, to provide diagnosis, treatment and support.

4.) **References:**

Choy RYS, Monro JA , Smith CW. Electrical Sensitivities in Allergy Patients. *Clinical Ecology* **4(3)**: 93-102, 1987.

Smith CW, Choy RYS, Monro JA. The Diagnosis and Therapy of Electrical Hypersensitivities. *Clinical Ecology* **6(4)**: 119-128, 1990.

Smith C.W. Nursing the Electrically Sensitive Patient, *Complementary therapies in nursing & midwifery* **3**, 111-116, 1997.

Notes:

THE DIAGNOSIS AND THERAPY OF EM HYPERSENSITIVITY

Cyril W. Smith Ph.D.

School of Acoustics and Electronics, University of Salford

Severely hypersensitive patients will have acquired inappropriate reactions to many chemical, environmental and nutritional substances at very low concentrations. If about 15% of any given population function to some extent below their best possible performance through some regulatory system defect, 1% of these will have added electrical hypersensitivity to their package of sensitivities. Frequencies represent "triggers" of hypersensitivity reactions (including heart sensitivities) to the electromagnetic environment. Reactions are critically dependent on frequency which may be anywhere from millihertz (MHz) to gigahertz (GHz) and beyond although, reactions may only appear following synergistic chemical stimulation. The symptoms observed are bizarre and wide-ranging and may cover any possible malfunction of the autonomic nervous system.

The first procedure for testing and treating electrically hypersensitive patients was based on the therapy for chemical and nutritional allergic responses developed by Dr. Joseph Miller. The patient was challenged in a clean environment with frequencies from electrical oscillators at a typical environmental intensity and the provoking and

neutralizing frequencies were observed. The latter were imprinted into water and used therapeutically as if an allergen dilution. This technique can only be used if the patient reacts within seconds and is not too sensitive. Patients who are extremely sensitive, who live at a distance, or who are unfit to travel, can imprint their body frequencies into the water by succussion which can then be mailed for measurement if wrapped in aluminum foil. This is a more patient-friendly method and has replaced the original procedure.

Neutralizing frequency imprints into water may alleviate symptoms and restore the normal endogenous electrical activity to a level at which it no longer perturbed by frequencies in the external environment. However, if there is an appreciable body load of toxic chemicals, their chemical frequency signatures may prevent effective treatment with frequency imprinted water until the chemicals are eliminated due to a "winner-takes-all" effect of the strongest signal, which may become imprinted too.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Richard P. Wedeen, M.D.** Friday, 11:00 a.m., June 9, 2000

SPEECH TITLE: **Lead and Hypertension: Who cares?**

The information below has been provided by the speaker.

1.) **Goals and objectives:** Learn about the evidence that lead causes hypertension.

2.) **Outline of talk/abstract:** Hypertensives with renal failure have significantly more chelatable lead than do hypertensives without renal failure. Since renal failure does not cause increased chelatable lead, these findings support the view that lead causes hypertension. In addition, blood lead predicts blood pressure within the "normal" ranges of both.

3.) **Conclusion of what is to be learned:** Lead contributes to hypertension in the general population. Experts in "essential hypertension" are more interested in treatment than prevention.

4.) **References:**

Wedeen, RP, Blood lead levels, dietary calcium, and hypertension(Editorial). Ann.Int.Med. 120:403-404,1985

Notes:

LEAD AND HYPERTENSION: WHO CARES?

Richard P. Wedeen, M.D.

Department of Veterans Affairs Health Care System, East Orange, NJ and The University of Medicine and Dentistry of New Jersey - New Jersey Medical School, Newark, NJ

The treatment of hypertension is an enormous medical cost in the United States. NHANES III records that 50 million Americans suffer from essential hypertension and hypertension is cited as the cause of end-stage renal disease in 28% of chronic dialysis patients. While the literature of occupational medicine, epidemiology and nephrology document an association between lead and hypertension, the literature on hypertension shows no awareness of this preventable cause. No attempt has been made to determine the contribution of lead to hypertensive disease.

The role of lead in the induction of hypertension is particularly relevant to male African-American population who are four-fold over-represented in end-stage renal disease programs in the United States and who are known to have the highest exposure to lead in childhood compared to other Americans. Hypertension and diabetic nephropathy are the major causes of end-stage renal disease in black Americans. And hypertension is the best predictor of end-stage renal disease in diabetics. NHANES II revealed that black male children have the highest blood levels in the United States. The possibility that lead exposure contributes to the excessive prevalence of hypertension and end-stage renal disease due to diabetic nephropathy in blacks has never been investigated.

Chronic interstitial nephritis induced by lead was first described in Queensland Australia at the turn of the century. The Australians noted that lead nephropathy was often associated with gout, as had Alfred Baring Garrod in his original description of the chemical basis of gout in 1859. Recognition of the association between lead and hypertension awaited the development of the sphygmomanometer at the end of the nineteenth century. The causal relationship between lead and high blood pressure was first explicitly stated by Theodore Janeway in 1912. Subsequent clinical studies have usually found an association between lead and hypertension when the question was specifically raised. In the 1980s, beginning with analyses of NHANES II data, reports from all over the world indicated that blood lead predicts blood pressure even when both measurements are within the normal range. Combined with evidence that neither renal failure nor hypertension per se cause elevated blood lead levels, the association between lead and blood pressure suggests that lead causes hypertension. The observation that hypertensives with renal failure and elevated lead stores had hypertension of shorter duration than hypertensives with renal failure but normal lead levels suggests that lead is responsible for both the hypertension and the renal failure. The appearance of renal arteriolar disease in occupational lead nephropathy before the development of hypertension suggests the possibility that renal micro vascular damage is the mechanism whereby lead induces hypertension. Essential hypertension might be designated lead nephropathy if appropriate diagnostic testing were undertaken. The failure to investigate these possibilities indicates that American physicians are more concerned with treatment than with disease prevention.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Kalpana D. Patel, MD** Friday, 11:30 a.m., June 9, 2000

SPEECH TITLE: **Effectiveness of Estrogen Replacement and Intravenous Magnesium Therapy to Correct Dyshomeostasis and Dysfunctioning Vascular Endothelium**

The information below has been provided by the speaker.

1.) **Goals and objectives:**

a) To demonstrate the effectiveness of estrogen HRT in perimenopausal women to relieve TIA and cerebrovascular disease having atypical manifestations.

b) To demonstrate efficacy of estrogen and magnesium to correct dyshomeostasis and dysfunctioning cerebral vascular endothelium in perimenopausal women.

c) To demonstrate reversal of symptoms of stroke and TIA with the use of estrogen replacement and intravenous magnesium therapy.

2.) **Outline of talk/abstract:**

3.) Conclusion of what is to be learned:

a) Treatment with estrogen hormone replacement therapy must be considered in every perimenopausal or menopausal female with symptoms of cerebrovascular dysfunction, i.e., TIA, transient strokes and migraine with aura etc.

b) Intravenous magnesium therapy is very effective for the reversal of cerebrovascular dysfunction and stroke in perimenopausal women in the early stage.

c) Vascular endothelial dysfunction can be effectively treated with estrogen hormone replacement and vascular tone modulators, i.e., magnesium, etc.

d) Age dependent degenerative changes and cerebrovascular accidents can be prevented with proper use of massive avoidance, environmental control, rotary diversified diet of organic food, glass bottled spring water, oral magnesium, nutrients, antioxidants and HRT by an astute clinician.

4.) **References:**

Cerebrovascular Disease and Stroke in Women. Cardiology 77, Supple 2:80-90, 1990

The Differential Effects of Estrogens and Progestins on Vascular Tone. Human Reproduction Update 5 (3):205-9, 1999 May-June. Comments in Human Reprod. Update 1999 (May-Jene 5 :189-90.

Estradiol Protects Against Ischemic Injury. Journal of Cerebral Blood Flow and Metabolism 18 (11):1253-8 1998 Nov.

Migrainous Visual Accompaniments Are Not Rare in Late Life. Stroke 29(8):1539-43 1998 August.

Ischemic Strokes and Use of Estrogen, Estrogen Program as Hormone Replacement. Stroke 29(1): 23-8 1998 Jan.

Pathogenesis of Migraine. Seminars in Neurology 17(4):335-41, 1997.

History of Migraine and Risk of Cerebral Ischemia in Young Adults. Lancet 347(9014):1503-6, 1996-June 1.

Notes:

EFFECTIVENESS OF ESTROGEN REPLACEMENT AND INTRAVENOUS MAGNESIUM THERAPY TO CORRECT DYSHOMEOSTASIS AND DYSFUNCTIONING VASCULAR ENDOTHELIUM

Kalpana D. Patel, MD, FAAP, FAAEM

Director of Allergy & Environmental Health Center - WNY

C.H., a 43 year old, white female, nulliparous with inhalant, food and chemical sensitivity, started having recurrent episodes of headaches, weakness and an abnormal sensation on the left side of her face, that started spreading to her left extremities. She started experiencing progressive weakness and parasthesia momentarily, also affecting her speech which became slurry; and shaking of right extremity on and off throughout the day for the last ten months. She also noticed fatigue, weakness, dizziness, myalgia, muscle spasms, shakiness, palpitation, depression, mood swings, PMS, inability to cope with stress and high blood pressure.

Past history of hysterectomy without removal of both ovaries.

She saw three physicians, including an internist, cardiologist and neurologist. She had a complete comprehensive work up, which was negative except the MRA which showed a small blood vessel vasculitis. She was on multiple medications, including Congentin for shaking of the extremities, Hydrochlorhiazide for hypertension, and Tegretol to suppress her neurological symptoms. She did not experience any relief of her symptoms and she was unable to tolerate them do to her chemical sensitivities; therefore, she discontinued them.

Due to the complex nature of her problems she came to see us for evaluation and treatment of her condition. Details will be discussed.

Following comprehensive evaluation she was placed on a treatment program, inclusive but not limited to, massive avoidance, environmental control, rotary diversified diet of organic foods, glass bottled spring water, injection treatment for the incitants to which she was found sensitive, nutritional supplements, antioxidants, intravenous infusions of magnesium, Vitamin C, B complex and other nutrients, chelation therapy to remove toxic heavy metals, estrogen patch for hormone replacement, thyroid supplements for hypothyroidism, and heat depuration-physical therapy program for chemical exposure. She has done extremely well on this program, and is overall 95% better in 90 days.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Jean Monro, MD** Friday, 1:30 p.m., June 9, 2000

SPEECH TITLE: **Heart Rate Variability in the Food and Chemically Sensitive Patient**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To illustrate that Coca's pulse test can be evaluated during testing.

2.) **Outline of talk/abstract:** Heart rate variability assessment before/during/after neutralization testing.

3.) **Conclusion of what is to be learned:** The relevance of pulse changes.

4.) **References:**

Sait JL, Wood AW, Sadafi HA, A study of heart rate and heart rate variability in human subjects exposed to occupational levels of 50 Hz circularly polarized magnetic fields; Mcd Phys 1999 Jun; 21 (5): 361-9.

Task Force of The European Society of Cardiology and The North American Society of Pacing and Electro physiology, Heart rate variability - Standards of measurement, physiological interpretation, and clinical use; Eur Heart J., Vol 17, March 1996; 354-381.

Notes:

HEART RATE VARIABILITY IN THE FOOD AND CHEMICALLY SENSITIVE PATIENT

Jean Monro, MD

Medical Director of Breakspear Hospital, England

GOALS AND OBJECTIVES: In the first presentation heart rate variability will be shown before, during and after neutralization testing. The second presentation will review and discuss heart rate variability with and without electromagnetic effects, to illustrate the dangers of electromagnetic fields.

BACKGROUND: One hundred years ago there were no domestic electrical devices. A century later, in the western world it is impossible to find people who do not live or work in an electrical "smog".

This electric pollution may cause a syndrome called "Electrical Sensitivity", and typical symptoms include headache, depression, muscular weakness, inco-ordination and even blackouts. These symptoms may be caused in electrically sensitive people by many types of electromagnetic radiation including radio and microwaves, infra-red, normal and ultra-violet light, x-rays, and cosmic radiation.

At present, there are few doctors and scientists who fully appreciate the fundamental importance of electromagnetic radiation as the cause of a wide variety of illnesses. More importantly, so few doctors understand that electromagnetic radiation may offer us a new way of curing many diseases and symptoms, previously unresponsive to treatments with present methods. Up until the present the principles of physics have been used almost solely for diagnostic rather than curative purposes. X-rays, ultrasound scans, nuclear magnetic resonance and CAT scans are invaluable in the diagnosis of disease, however, it is predicted that in the 21st century electromagnetic radiation will be used for its untapped potential as a curative gent.

As a general rule the most dangerous devices are high current devices and those that require a very strong magnetic field for their operations like motors and transformers. Electrical sensitivity affects at least 1 in 1,000 of the population. Almost all electrically sensitive people are also sufferers from food and/or chemical allergies.

Non-ionising electromagnetic radiation is able to cause a wide variety of symptoms especially those related to blood vessels and the brain and nerves. These symptoms include flushing, blushing, palpitations, diarrhea, muscular aches, pins and needles especially in the hands and feet, dizziness, fits and blackouts, disorientation, headaches, noises in the head, depression and suicide, and persistent tiredness unrelieved by rest. Electrical sensitivity may also mimic neurological diseases such as paralysis, epilepsy and multiple sclerosis. Other diseases and symptoms associated with prolonged exposure to magnetic and electric fields include headache, depression, suicide, miscarriage, cancer and leukemia.

The reasons that electricity is often poorly recognized as a cause of illness is because of the fact that there is often a long latent period, 3-5 years, between exposure to the current and the effect being noticed. In addition, there may be a further 10 years before a well defined disease shows itself.

The human body is highly electrically active. Minute currents can be measured from every cell in the body and individual organs such as the heart and brain are routinely monitored to assess disease. Electrical activity is absolutely fundamental to life and the state of health of the body can be tested electrically in several ways. One technique that was developed in Russia is Kirlian photography. In this technique a very high frequency electric current is applied to the body and that part photographed. The electrical discharge given off changes according to the state of health.

A technique has been to expose electrically sensitive patients to very weak non-ionising electromagnetic radiation. The frequency of the electromagnetic radiation can be very accurately controlled by using crystal controlled oscillators. This testing can be done double-blind, and in many hundreds of cases we found that an electrically sensitive patient may be ill using one frequency but can be healed by using a second frequency.

Our work at Breakspear Hospital has found that there is a very strong similarity between electrically sensitive patients' frequencies by using an electrical oscillator, and duplicating the effect by giving patients provocation/neutralization testing. It is believed that the provocation/neutralizing testing does not work by rules of pharmacology or

chemistry since the dilution of many solutions is so great that there should not be a single molecule of the original substance contained in the injection.

Another method of testing a patient's response to different frequencies is to use the technique of applied kinesiology.

Electrical fields can be screened by using an earthed Gaussian screen, also called a Faraday Cage. This screening may be carried out relative cheaply by papering the walls of rooms with ordinary aluminum foil which is then connected to earth.

A similar effect may be obtained by using a material which has been sprayed with silver which then conducts electricity. This material may even be made into clothes.

Unfortunately, it is very difficult to screen magnetic fields but individual devices can be greatly modified to reduce their electric and magnetic radiation. For example, we can advise you about low radiation computer monitors which give out one tenth of the level amount of radiation of standard devices.

By understanding the principles of biophysics it has been possible to treat a large number of patients sensitive to non-ionising radiation. We have seen patients who have become very ill when they have lived close to high voltage powerlines due to their exposure to 50 Hz. When they moved away from this powerful radiation their health improved and electrical sensitivity disappeared. We have seen people who have used personal radio transmitters such as mobile telephones and deaf aids transmitter who have become unable to work due to the radio transmitter making them feel tired all the time. We have had patients sensitive to marine and aeronautical radar frequencies who become ill when living near airports and coastal marine radar facilities.

Finally, we have had patients becoming ill due to the electrical oscillators found in their computers and quartz watches.

Treatment of all these cases has consisted of reducing the Total Load of allergic problem in air, food, water and also the Total Load of non-ionising electromagnetic radiation from all sources.

Provocation/neutralization injections can also be helpful to find the healing frequency for an individual patient and the patient may be given their frequency by holding a glass vial containing water energized at that frequency.

CONCLUSION: The relevance of pulse changes in people will be highlighted, and the benefits of electromagnetic screening will be outlined.

HEART RATE VARIABILITY IN THE FOOD AND CHEMICALLY SENSITIVE PATIENT

Presented by

Dr Jean Monro

MB BS MRCS LRCP FAAEM DIBEM MACOEM

Medical Director
Breakspear Hospital
England

HEART RATE VARIABILITY:

The last two decades have witnessed the recognition of a significant relationship between the autonomic nervous system and cardiovascular mortality, including sudden cardiac death. Experimental evidence for an association between a propensity for lethal arrhythmias and signs of either increased sympathetic or reduced vagal activity has encouraged the development of quantitative markers or autonomic activity.

Heart rate variability (HRV) represents one of the most promising such markers. The apparently easy derivation of this measure has popularized its use. As many commercial devices now provide automated measurement of HRV, the cardiologist has been provided with a seemingly simple tool for both research and clinical studies.

Heart rate variability has considerable potential to assess the role of autonomic nervous system fluctuations in normal healthy individuals and in patients with various cardiovascular and non-cardiovascular disorders. HRV studies should enhance our understanding of physiological phenomena, the actions of medications, and disease mechanisms.

ABSTRACT INFORMATION & NOTES

Speakers Name: Stephen A. Levine, Ph.D. Friday, 2:00 p.m., June 9, 2000

SPEECH TITLE: Antioxidant Adaptation, Cancer and Cardiovascular Disease

The information below has been provided by the speaker.

1.) **Goals and objectives:** To understand underlying biochemical redox mechanisms effective for treating cancer & optimizing health

2.) **Outline of talk/abstract:** Basic free radical chemistry discussed, types of stress-cancer causation attributed to stress, due to electrical imbalances, free radical excess & oxygen deficit. Treatment options obvious from understanding.

3.) **Conclusion of what is to be learned:** Treatment modalities will be presented along w/ conceptual frame work of free radical/antioxidant mechanisms in health and disease

4.) **References:** Textbook: ANTIOXIDANT ADAPTATION - SA Levine & PM Kidd, 1984

SA Levine, Organic Germanium text, Suzuki/F.Pollard RB, Induction of interferon Microbial II Immunol 1985 29(1):65-74, "Cancer & Anemia" Focus- ARG Newsletter, 1999.

Notes:

ANTIOXIDANT ADAPTATION, CANCER AND CARDIOVASCULAR DISEASE

Stephen A. Levine, Ph.D.

I. Antioxidant Adaptation & Beyond

In healthy cells and tissues, free radicals produced by oxidative phosphorylation, biological oxidations, and chemical and drug detoxification reactions are normally insulated from susceptible molecules and enzymes by cell membrane barriers that contain numerous antioxidant molecular species derived from nutrients. Such antioxidant molecules function in concert with protective antioxidant enzyme systems to maintain optimal cellular redox balance - a healthy balance between oxidative stress and the antioxidant defense capacity of the cell. Any substantial shift in the local oxidation-reduction balance in response to chemical oxidant exposure, intensified endogenous generation of oxidant molecular species, physical trauma, or infection can directly affect the viability and functioning of the cell, tissue, or organ system. A patient's health is, therefore, related to the additive oxidative stress imposed on the individual's antioxidant defense system by exogenous and endogenous oxidant stressors.

The antioxidant adaptation hypothesis is well supported by findings from some of the best-studied human congenital metabolic diseases. It offers a rational biochemical interpretation for certain clinical phenomena in food and chemical hypersensitive patients. The antioxidant biochemical adaptation theory is based on the most recently elaborated principles of electronic biochemistry which underlie chemical toxicology and carcinogenesis. Inherent in this theory is the assumption that the myriad of factors that precipitate oxidative stress operate through avenues involving direct tissue damage by free radicals and other activated oxygen species. This theory, and its further implications, are also strongly supported by the extraordinarily broad therapeutic potential attributed to key antioxidant nutrient factors such as beta-carotene, vitamins A, C, and E, selenium, and zinc.

II. Beyond Antioxidant Adaptation in Cancer & Disease

When the body goes beyond antioxidant adaptation, it can no longer defend itself against stress (cancer being a good example of this). When pushed beyond its limits (beyond adaptation), the antioxidant system, which is a sophisticated system of aerobic metabolism, reverts to a primitive system of anaerobic metabolism, leaving the body open to disease.

It is my proposal that cancer cells do not have the antioxidant adaptation capacity. They have gone beyond the ability to adapt to oxidative stress. And this in part characterizes their transformed state.

Oxidative theories and treatment approaches for cancer and disease will be discussed. Also to be discussed, will be the main categories of stress, which play a key role in exhausting the body's antioxidant defense system.

III. Beyond Antioxidant Adaptation in Cardiovascular Disease

The function of the cardiovascular system is to facilitate the delivery of oxygen and nutrients for the purpose of cellular respiration. Consistent with other degenerative diseases, lipid peroxidation and free radical processes mediate deterioration in cardiovascular disease, and eventual hypoxia results in cellular damage perceived through free radical lipid peroxidation and vascular injury. More on this subject will be discussed.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Sherry S. Rogers, M.D.** Friday, 2:30 p.m., June 9, 2000

SPEECH TITLE: **The Environmental Medicine Approach to Solving Recalcitrant Cardiology Conditions**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To teach the physician how to work through the total load of the most common causes of heart disease

2.) **Outline of talk/abstract:** Case examples will demonstrate how to improve end-stage congestive heart failure, angina and cardiac arrhythmias that had failed all that medicine could offer.

3.) **Conclusion of what is to be learned:** Physicians should begin to understand that they have more healing power by identifying the underlying causes of cardiac disease than merely masking symptoms with drugs.

4.) **References:**

1. Ornish D, Scherwitz, LW, Brand RJ, et al, Intensive lifestyle changes for reversal of coronary heart disease, J Amer Med Assoc 1998;289:2001-2007
2. Drexler H, Aeiner AM, Meinzer K, Lust H, Correction of endothelial dysfunction in coronary microcirculation of hypercholesterolaemic patients by l-arginine, Lancet 338:1546-50,1991

3. Baggio E, Gandini R, Plancher AC, Passeri M, Carmosino G, Italian multicanter study on the safety and effecacy of coenzyme Q10 as adjunctive therapy in heart failure. CoQ10 Drug Surveillance Investigators, Mol Aspects Med, 1994;15 Supple: S287-94
4. Msumura Y, Kobayaski A, Yamazaki N, Myocardial free carnitine and fatty acylcarnitine levels in patients with chronic heart failure, Jap Circul J, 54; Dec 1990,1471-1476

Notes:

THE ENVIRONMENTAL MEDICINE APPROACH TO SOLVING RECALCITRANT CARDIOLOGY CONDITIONS

Sherry A. Rogers, M.D.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Garth L. Nicolson** Friday, 1:30 p.m., June 9, 2000

SPEECH TITLE: **Diagnosis and Treatment of Cell-Invasive Bacterial Infections in Chronic Fatigue Syndrome (ME), Fibromyalgia Syndrome,Gulf War Illnesses and Rheumatoid Arthritis**

The information below has been provided by the speaker.

1.) **Goals and objectives:**

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:**

4.) **References:**

[1] Nicolson GL, Nasralla M, Haier J, Erwin R, Nicolson NL, Ngwenya R. Mycoplasmal infections in chronic illnesses: Fibromyalgia and Chronic Fatigue Syndromes, Gulf War Illness, HIV-AIDS and Rheumatoid Arthritis. Med Sentinel 1999; 4:172-176.

[2] Nicolson GL, Nasralla M, Nicolson NL. The pathogenesis and treatment of mycoplasmal infections. Antimicrob Infect Dis Newsl (Elsevier Press) 1999; 17(11):81-88

[3] Nicolson GL, Nicolson NL. Chronic fatigue illness and Operation Desert Storm. J. Occup Environ Med 1996; 38:14-16

[4] Nicolson GL, Nicolson NL. Doxycycline treatment and Desert Storm JAMA 1996; 273:618-619

[5]. Nicolson GL, Considerations when undergoing treatment for chronic infections found in Chronic Fatigue Syndrome, Fibromyalgia Syndrome and Gulf War Illnesses. (Part 1). Antibiotics Recommended when indicated for treatment of Gulf War Illness/CFS/FMS (Part 2). Intern J Med 1998; 1:115-117, 123-128.

[6] Nicolson GL, Nicolson NL, Nasralla M. Mycoplasmal infections and Chronic Fatigue Illness (Gulf War Illness) associated with deployment to Operation Desert Storm. Intern J Med 1998; 1:80-92.

[[7] Nicolson GL, Nicolson NL. Diagnosis and treatment of mycoplasmal infections in Persian Gulf War Illness-CFIDS patients. Int J Occup Med Immunol Tox 1996; 5:69-78

[8] Nicolson GL, Nasralla M, Haier J, Nicolson NL. Diagnosis and treatment of chronic mycoplasmal infections in Fibromyalgia Syndrome and Chronic Fatigue Syndrome: Relationship to Gulf War Illness. Biomed Therapy 1998; 16:266-271

[9] Nasralla, M., Haier, J. and Nicolson, G.L. Multiple mycoplasmal infections detected in blood of Chronic Fatigue and Fibromyalgia Syndrome patients. Eur J. Clin Microbiol Infect Dis 1999; 18:859-865.

[10] Haier J. Nasralla M, Franco AR, Nicolson GL. Detection of mycoplasmal infections in blood of patients with Rheumatoid Arthritis. Rheumatol 1999; 38:504-509.

[11] Nicolson GL, Nicolson NL. Gulf War Illnesses: complex medical, scientific and political paradox. Medicine Conflict & Survival 1998: 14: 156-165.

[12] Nicolson GL, Nasralla M, Haier J, Nicolson NL. Gulf War Illnesses: Role of chemical radiological and biological exposures. In: War and Health, H. Tapanainen, ed., Zed Press, Helinsiki, 2000; in press.

[13] Nicolson GL, Nasralla M, Franco AR, Erwin R, Nicolson NL, Ngwenya R, Berns P. Diagnosis and integrative treatment of intracellular bacterial infections in Chronic Fatigue and Fibromyalgia Syndromes, Gulf War Illness, Rheumatoid Arthritis and other chronic illnesses. Clin Pract Alt Med 2000; in press.

Notes:

DIAGNOSIS AND TREATMENT OF CELL-INVASIVE BACTERIAL INFECTIONS IN CHRONIC FATIGUE SYNDROME (ME), FIBROMYALGIA SYNDROME,GULF WAR ILLNESSES AND RHEUMATOID ARTHRITIS

Garth L. Nicolson, Marwan Nasralla, Joerg Haier and Nancy L. Nicolson

Patiens with chronic illnesses, such as Chronic Fatigue Syndrome (CFS/ME), Fibromyalgia Syndrome (FMS), Gulf War Illness (GWI) or Rheumatoid Arthritis (RA), often have overlapping chronic signs and symptoms. Although chronic illness often start

with an acute episode involving exposures to chemicals, allergens, viruses, etc., eventually major sources of chronic morbidity are various viral, bacterial and fungal secondary infections [1,2]. GWI, CFS/ME and FMS patients slowly present with complex, multi-organ signs and symptoms, such as polyarthralgia, chronic fatigue, short-term memory loss, sleep difficulties, headaches, intermittent fevers, skin rashes, diarrhea, vision problems, nausea, breathing and heart and other problems. Although there is not yet a case definition for some of these chronic illness, such as GWI, the signs and symptoms of GWI loosely fit those of CFS/ME and FMS [3]. Nucleoprotein Gene Tracking (NGT) and Forensic Polymerase PCR showed that 45% (NGT) and 50% (FPCR) of 200 GWI patients and their immediate symptomatic family members had mycoplasmal infections inside their blood leukocytes. The most common species (>80%) found was M. *fermentans.* In contrast, in nondeployed, healthy adults the incidence of mycoplasma-positive tests were 0-6%. Mycoplasma-positive cases of GWI/CFS/ME have been successfully treated with multiple 6-week cycles of antibiotic [4,5] plus nutritional support [4]. After up to 6 cycles of therapy, 69/87 recovered and 18/87 were still undergoing therapy [6,7]. GWI patients who recovered from their illness after several (3-7) 6-week cycles of antibiotic therapy were retested for evidence of mycoplasmal infections and were found to have reverted to a mycoplasma - negative phenotype, and they were less environmentally sensitive [6,7]. The results were compared to 203 patients with CFS/ME or FMS and 100 patients with RA. Using FPCR 144/203 CFS/ME/FMS patients were positive for mycoplasmal infections (60%), whereas only 7/92 healthy controls were positive [8]. In mycoplasma-positive CFS/ME/FMS patients were found a variety of mycoplasma species (primarily *M. fermentans, M. hominis, M. pneumonia and M. penetrans*) [8.9]. RA patients also had high frequencies of mycoplasmal infections (45%), mostly multiple infections, and these patients also had various species of mycoplasmas in their blood leukocyte fractions [10]. We conclude that subsets of GWI, CFS/ME, FMS and RA patients have mycoplasmal and often other transmittable chronic bacterial, viral and fungal infections as well, and treatment of these chronic patients with appropriate antibiotics plus nutritional support can result in slow recovery from their conditions [1,5-7]. We propose that GWI is to a large degree due to multiple exposures to chemical, radiological and biological agents that cause multifactorial illnesses, some of which can be transmittable to immediate family members and involve chronic infections [11,12]. Civilians with CFS/ME, FMS or RA also often show the presence of multiple chronic bacterial infections (*Mycoplasma, Rickettsia, Brucella, Borrelia* and others), and these patients can be successfully treated similar to GWI patients with long-term antibiotics, oxidative therapy and nutritional supplements and other support [13]. (Further information and publications see: www.immed.org).

ABSTRACT INFORMATION & NOTES

Speakers Name: **Nancy Didricksen, Ph.D.** Friday, 4:00 p.m., June 9, 2000

SPEECH TITLE: **The Use of the Halstead-Reitan Neuropsychological Test Battery to Measure Neurotoxic Effects in Chemically-Exposed Individuals**

The information below has been provided by the speaker.

1.) **Goals and objectives:**

1. Describe the effects of neurotoxic exposure on neurocognitive functioning.

2. Describe the use of the Halstead-Reitan Neuropsychological Test Battery to evaluate neurocognitive dysfunction in chemically-exposed individuals.

3. Report the analysis of data of the various components of the Halstead-Reitan Neuropsychological Test Battery.

4. Suggest directions for future research and assessment recommendations.

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:** The Halstead-Reitan Neuropsychological Test Battery is an appropriate and useful method of measuring the effects of neurotoxic exposure as part of a comprehensive neuropsychological evaluation. The specific components of the HRB which are most sensitive to dysfunction in neurotoxically-exposed individuals are useful as components of a brief neuropsychological evaluation in addition to the Comprehensive Neuropsychological Screen.

4.) **References:**

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Notes:

THE USE OF THE HALSTEAD-REITAN NEUROPSYCHOLOGICAL TEST BATTERY TO MEASURE NEUROTOXIC EFFECTS IN CHEMICALLY-EXPOSED INDIVIDUALS

Nancy Didricksen, Ph.D.

The major classes of neurotoxic chemicals are organic solvents, pesticides (e.g., organophosphates and organochlorines) and heavy metals (e.g., lead, mercury). However, many other substances can adversely affect neuropsychological functioning as well as personality and behavioral functioning. More than 850 chemicals have been identified as having neurotoxic effects which may be subtle. Deterioration may be gradual and often insidious.

Many neurotoxic substances produce adverse changes in attention, concentration, learning and memory, comprehension, abstract-reasoning ability, and problem-solving, as well as in sensory and motor functioning. Patients report absentmindedness, disorientation, mental slowing, intellectual inefficiency, balance and coordination problems, and impaired performance of work-related duties.

The Halstead-Reitan Neuropsychological Test Battery for Adults was administered to 94 patients who have been exposed to a variety of neurotoxic substances as part of a comprehensive assessment of neurotoxic effects on cognitive functioning. The HRB, a fixed, rather than flexible battery, is the most thoroughly balidated and researched neuropsychological test battery in the country and measures a wide range of behavioral functions suserved by the brain. Test results may be used to make clinical inferences regarding cerebral damage.

The data of the 94 patients who had undergone comprehensive evaluation were examined to determine: (1) The degree of neurocognitive dysfunction after neurotoxic exposure, (2) primary areas of dysfunction, (3) lateralization or localization effects, (4) pathognomonic signs, (5) patterns in central processing, and (6) the degree of correlation with the Comprehensive Neuropsychological Screen which was developed on neurotoxically-exposed individuals. Results suggest directions for future research as well as recommendations for efficient assessment of toxically-exposed individuals.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Tonya G. Callaway** Friday, 1:30 p.m., June 9, 2000

SPEECH TITLE: **Clinical Analysis Questionnaire (CAQ): Psychological Profiles of Neurotoxically-Exposed Patients**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To present recent findings concerning the overall CAQ psychological profiles of a sample of neurotoxically-exposed (NE) patients in comparison to the CAQ normal adult standardization group and the CAQ clinical population. A brief synopsis of the following topics will also be provided: the demographic characteristics of the sample; the CAQ and what it measures; some of the limitations involved in the psychological evaluation of NE patients and ; the need for careful interpretation of psychometric data in neurotoxic exposure cases.

2.) **Outline of talk/abstract:**

- Psychological Assessment in Neurotoxicity Cases
- The CAQ and What the CAQ Measures
- Purpose of the Present Study
- Overview of the Demographic Characteristics of the NE patients
- Synopsis of Results and Relevant Conclusions

3.) **Conclusion of what is to be learned:** Present results suggest that the personality functioning of this NE sample is not similar to those with psychiatric diagnoses or to normal healthy adults. These findings do not lend support to the supposition that patients experiencing neurotoxic-related morbidity are malingers or have an underlying primary psychological disorder.

4.) **References:**

1. Fielder, N. (1996). Neuropsychological approaches for the detection and evaluation of toxic symptoms. Environmental Health Perspectives, 104(Suppl 2), 239-245.
2. Fielder, N., Feldman, R.G., Jacobson, J., Rahill, A., & Wetherell, A. (1996). The assessment of neurobehavioral toxicity: SGOMSEC joint report. Environmental Health Perspectives, 104(Suppl. 2), 179-191.
3. Hartman, D.E. (1995). Neuropsychological toxicology: Identification and assessment of human neurotoxic syndromes (2nd ed.). New York: Plenum Press.
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Notes:

CLINICAL ANALYSIS QUESTIONNAIRE (CAQ): PSYCHOLOGICAL PROFILES OF NEUROTOXICALLY-EXPOSED PATIENTS

Tonya G. Callaway

Exposure to neurotoxicants can lead to the development of psychological symptomatology in humans. Personality changes are often not affiliated with neurotoxic

exposure; referrals to mental health professionals are prevalent among the neurotoxically-ill. Prior assessment research has indicated high levels of depression, hypochondriasis, anxiety, and disturbed thinking amid this patient population. Findings appear to support claims that such symptoms are a reflection of an underlying psychological disorder. However, there are limitations involved in the psychological evaluation of these patients: appropriate comparison norms are non-existent and; mental health care providers are typically not trained in neurotoxicology. This study extended previous psychological assessment findings in neurotoxic exposure cases using an instrument, the Clinical Analysis Questionnaire (CAQ), lacking in prior published research protocols. The overall aim of this research was to determine the personality profiles of 162 (82 women, 80 men) neurotoxically-exposed (NE) patients. Subjects ranged in age from 16 to 69 (M = 44.59). 53% of the participants had no previous psychological/psychiatric history. The CAQ is a 272-item self-report questionnaire that assesses both normal personality traits (yielding 16 scales) and pathological personality characteristics (yielding 12 scales). Resulting CAQ sten (i.e., standard ten transformed scores) profiles demonstrated the hypochondriasis scale (D1) was significantly elevated in both neurotoxic exposure groups (i.e., males, females) relative to the applicable CAQ normal adult standardization groups. NE males elevated the anxious depression scale (D4) as well. These findings are not surprising given that NE patients often report experiencing multiple physical and cognitive symptoms. Additionally, the resulting NE group profile (i.e., mean raw score) was compared to that of the CAQ normative clinical population. Sizable variations were noted between the 2 groups across normal personality traits and psychopathology factors. Of interest, the NE group clinical profile did not resemble that of the psychiatric population on 9 of 12 clinical scales. Results suggest the NE sample is not representative of a group with primary psychological diagnoses. The overall NE profile (i.e., mean raw score) was also compared to that of the CAQ normal adult population. The NE group differed significantly from the general population norms across a number of personality dimensions (i.e., 25 of 28 scales). Of note, the NE sample scored significantly higher on the Conformity (G) and Self-Sufficiency (Q2) scales. These results suggest that the NE patients tended to be: dominated by a sense of duty, exacting in character, conscientious, independent, and accustomed to taking action on their own. Personality traits such as these are not consistent with a profile of patients who tend to malinger. Overall, present results indicate that the personality functioning of this NE sample is not similar to those with psychiatric diagnoses or to normal healthy adults. Current findings do not support previous claims that patients experiencing neurotoxic-related morbidity represent a population of malingers or those with an underlying primary psychological disorder. Follow-up studies are needed. Of interest, a chronically-ill medical population should serve as a reference to ascertain if the NE group CAQ profile would resemble that of medical patients.

Saturday Session

8:15 ANNOUNCEMENTS/MODERATOR: Jean Monro, M.D.
8:30 "Heart Variability In Russian Sailors", Alexander Riftine, Ph.D.
8:50 Q & A

9:00 "Occupational Renal Diseases", Richard P. Wedeen, M.D.
9:20 Q & A
9:30 "Chemical Sensitivity Patients in Japan, Part I", Satoshi Ishikawa, M.D.
9:50 Q & A
10:00 BREAK WITH EXHIBITORS

MODERATOR: William Meggs, M.D.
10:30 "EM Fields in Health, in Therapies, as Hazards", Cyril W. Smith, Ph.D.
10:50 Q & A
11:00 The Environmental Aspects of Cardiovascular Disease, Part II", William J. Rea M.D.
11:20 Q & A
11:30 "Cardiovascular Nuclear Scanning", Theodore R. Simon, M.D.
11:50 Q & A
12:00n OPEN LUNCH

MODERATOR: Theodore Simon, M.D.
1:30 "Laser Acupuncture & Therapy in Chemical Sensitivity Patients", Tom Croley, Ph.D.
1:50 Q & A
2:00 "Allergy and the Heart", William Meggs, M.D.
2:20 Q & A
2:30 "Pesticide Poisoning - The Missed Diagnosis", Allan Lieberman, M.D.
2:50 Q & A

3:00 BREAK WITH EXHIBITORS
3:30 "Update on Clinical Aspects of Homocysteine", Jon Pangborn, Ph.D.
3:50 Q & A
4:00 "The Secret Life of Compost", Malcom Beck
4:20 Q & A
4:30 CASE STUDIES & PANEL/ MODERATOR: Allan Lieberman, M.D.
Case # 1EHC-D, David Hickey, M.D.
Case # 2 Tom Croley Ph.D.
Case # 3 William J. Rea, M.D.

6:00 RECEPTION WITH THE EXHIBITORS

ABSTRACT INFORMATION & NOTES

Speakers Name: **Alexander Riftine, Ph.D.** Saturday, 8:30 a.m. June 10, 2000

SPEECH TITLE: **Clinical Applications and Practical use of Fully Automatic System for Quantitative Assessment of Autonomic Nervous System Condition Based on Heart rate variability Analysis**

The information below has been provided by the speaker.

1.) **Goals and objectives:**

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:**

4.) **References:**

Notes:

CLINICAL APPLICATIONS AND PRACTICAL USE OF FULLY AUTOMATIC SYSTEM FOR QUANTITATIVE ASSESSMENT OF AUTONOMIC NERVOUS SYSTEM CONDITION BASED ON HEART RATE VARIABILITY ANALYSIS

Alexander Riftine, Ph.D.

Heart Rhythm Instruments, Inc.

Generally the method of Heart Rate Variability (HRV) gives us an ability to detect the early signs of the development of pathological processes or the presence of some functional disorders, which may no be revealed by the procedures of an ordinary physicla examination. Since the main regulation mechanism in Heart Rate Variability is that of an autonomic regulation, it is well known that autonomic response is the first response of any intervention to the organism or any physical, physiological, or psycho-emotional activity. Any pathological process will immediately provoke an ANS response.

In the analysis of HRV the Nerve-Express uses an effective visual representation called ***rhythmography*** which reflects the wave structure of HRV and serves as a "fingerprint" of the regulatory mechanisms. The spectral analysis of this wave "portrait" allows Nerve-Express to identify two main spectral components - High frequency (0.5-0.15 Hz) and Low frequency (0.15 - 0.04Hz). It has been found that there is a high correlation between power of the High frequency spectrum and the Tone of Parasympathetic Nervous System (PSNS) and between the Power of Low Frequency spectrum and the Tone of Sympathetic Nervous System (SNS). Nerve Express uses an algorithm which analyzes the peak amplitudes in both high and low spectral frequencies and can graph the relationship between the activity of SNS and PSNS. Nerve-Express uses a principally new classification of ANS showing 56 different states of autonomic condition, arranged into eight categories:

1. PSNS prevalence with average level of SNS activity: This category represents PSNS dominance. This state is usually noted when a patient is resting or the first stage fo sleep (specifically, dreamless sleep). In the second stage of slot SNS activity is generally

incresed, at times markedly thus, htis category is differentiated into four grades, dependent on the PSNS. This category somewhat limited, since it can only be noted in patients with strictly median values of SNS activity.

2. Increase in PSNS and SNS activity: This category is subdivided into sixteen different possible combinations of PSNS/SNS activity. It is characteristic of mostly healthy subjects.

3. SNS Prevalence: This category represents an increase in SNS compared to a median value of the PSNS. From the medicophysiological standpoin, this category represents a transitional stage between the second and the fourth categories.

NOTE: Categories 1 through 3 represent basically healthy persons but we have to keep in mind that healthy people may have two different physiological states. One state has a low level of sympathetic activity and the other has a significant increase of sympathetic activity. Both states are distinguished by an increase in parasympathetic activity. In stress theory by Salier stress is subdivided into two parts, stress and distress. It is understood that stress if reflected by an increase of PSNS and significant increase of SNS while distress is reflected by decrease of PSNS and significant increase of SNS. Conditions of healthy persons with significant increase of SNS and increase in PSNS are thus correspond to the Salier's ideao fo stress. For example this type of stress can be compared to whan an athlete feels befor competition.

4. PSNS decrease with SNS increase: This category can apply to both clinically sick, and clinically healthy individuals (defined as those not requiring hopitalization). However, the use of the term "healthy is not always appropriate since functional imbalance, from stress, physical exhastiion, nervous tension, infections, intoxication (including drug or alcohol), exacerbation of chronic conditions, and many other conditions may still be present. In such cases a decrease in PSNS DUE TO DEPRESSED psns NERVE CENTER CNA BE OBSERVED, ALONG WITH A SIMULTANEOUS Sympatho-ADRENERGIC activation, which is triggered by the struggle of the nervous system to balance itself.

NOTE: The Sailier's idea of distress correlates to Category 4(see Fig.2) where significant decrease of PSNS correspons to significant increase of SNS.

5. PSNS decrease with average level SNS: This category like the third, is transitional. Everything that pertains to the fourth category can be related to it, but here, the SNS activity is within median values. This means that stress, exhaustion, or nervous overload are unlikely. This category may often reflect a depression in the receptor system of the PSNS, indicating the possibility of chronic pathology.

6. SNS and PSNS decrease: The sixth category, especially at point 3 and 4, reflects a general involutionary degeneration of both the SNS and PSNS nervous centers. The majority of cases found in this category are either very old patients or those with diseases which cause a significant decrease in the sensitivity of the entire receptor system along with a partial degeneration of nervous centers. Examples are cancer patients, patients

after infarct of the myocard and any other disease which cause similar depression of the ANS centers.

NOTE: Point 6(1) of this category is an exception to this. It represents an insignificant, general decrease in ANS and approximates the point of Autonomic Balance. It can be interpreted as a border line value of Autonomic Balance.

7. Autonomic Balance: It is a category, even though formally it is only a point, and all other points in its vicinity that belong to the other six categories can be interpreted as border line values of the Autonomic Balance.

8. Slight increase in PSNS with decrease in SNS: The eighth category is rather unusual because normally an increase in PSNS is accompanied by an increase in SNS. This situation is observed in two cases: it is found in persons with special heart training for deep sea diving and it is found in patients with hyperkalemia or excessive levels of potassium ions which alters the usual polarized state of the cardiac muscle fibers and the result is a decrease in the rate and force of contractions. In fact, if the potassium ion concentration is very high, the conduction of cardiac impulses may be blocked and heart action may suddenly stop (cardiac arrest).

The methodology of use of the Nerve-Express is to compare ANS condition before and after any treatment or activity. For example, measurements of HRV taken before and after Neural Therapy showed the increase of myocard adaptability through additional analysis of transition period between supine and upright positions (orthostatic test).

For other therapeutic measures such as acupuncture and electro-therapeutics same methodology applies. In all cases, the target area for ANS condition, after successful completion of therapy, should be the increase of PSNS with normalization of SNS and reduction of discrepancy between supine and upright measurements.

The Nerve-Express was a subject of comparison study with the Chronos system (es. Gold standard) conducted by J.T. Bigger, Jr. MD at Columbia University. The purpose of the study was to validate Nerve Express as a reliable office system for purposes of assessment of risk in cardiovascular disorders, assessment of physical fitness, documentation of benefit for cardiac, chiropractic, and orthopedic rehabilitation. The results of this study showed excellent agreement between the two algorithms which suggests that the Nerve Express algorithms can predict death in coronary heart disease and can assess level of physical fitness.

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ABSTRACT INFORMATION & NOTES

Speakers Name: **Richard P. Wedeen, M.D.** Saturday, 9:00 a.m., June 10, 2000

SPEECH TITLE: **Occupational Renal Diseases**

The information below has been provided by the speaker.

1.) **Goals and objectives:** Learn about those occupational exposures which cause renal disease.

2.) **Outline of talk/abstract:** The occupational renal diseases are due to lead, mercury, cadmium, Chromium, uranium, germanium, silica, and arganic solvents.

3.) **Conclusion of what is to be learned:** Occupational diseases due to relatively high exposure in a few workers provide a model for detecting environmental renal diseases in while populations exposed to lower levels of these toxins.

4.) **References:**

Wedeen, RP, Renal and urinary tract disorders. In: Occupational Health: Recognizing and Preventing Work-Related Disease and Injury. B. Levy and D.W. Wegman(eds.) Philadelphia. Lippincott Williams and Wilkens, 1999.pp 641-52.

Notes:

OCCUPATIONAL RENAL DISEASES

Richard P. Wedeen, M.D.

Department of Veterans Affairs Health Care System, East Orange, NJ, and The University of Medicine and Dentistry of New Jersey - New Jersey Medical School, Newark, NJ

Occupational renal diseases due to high exposure of relatively few individuals to toxins in the workplace are important because they are preventable and because they provide models for understanding the consequences of low-level environmental exposure to large populations. Agents known to be responsible for chronic renal disease include heavy metals and organic chemicals. While the heavy metals regularly cause acute tubular necrosis when injected intravenously, lead and cadmium occasionally induce chronic

interstitial nephritis in individuals exposed heavily in the workplace over many years. The chronic interstitial nephritis caused by long-term occupational exposure to lead must be differentiated from the transient acute proximal tubule reabsorptive defect (Franconi syndrome) caused by the acute lead poisoning. Hypertension and gout frequently accompany occupational lead nephropathy but are absent from cadmium-induced renal disease which is characterized by calcium wasting, osteomalacia, and kidney stones. Minute dose of mercury can cause glomerular disease manifested as the nephrotic syndrome in sensitive individuals. Mercury, arsenic, chromium and uranium may leave residual chronic interstitial nephritis in victims who survive acute tubular necrosis, but chronic renal disease from occupational exposure to these metals has not been described. Organic compounds such as carbon tetrachloride can also produce acute tubular necrosis when inhaled in large dosage. Long-term low-level inhalation of industrial solvents has been found to cause a variety of glomerular diseases in numerous case control studies but these findings remain controversial because of the potential of recall bias, the complexity of the solvent mixtures, and the absence of accurate estimates of exposure. The finding of eosinophilic lysosomal bodies in the cells of proximal tubules in rats exposed to petroleum products (light hydrocarbon nephropathy) has not been associated with the development of chronic renal disease. Occupational exposure to silica has been identified as a cause of anti-neutrophile cytoplasmic antibody (ANCA) positive Wegener's granulomatosis. This immunologically-mediated vasculitis of lungs and kidneys represents an unusual manifestation of exposure to silica and may be present in the absence of pulmonary silicosis. Glomerulonephritis may also occur in association with the intense immunological manifestations of silicoproteinosis in the presence of severe silicosis.

Over 250,000 Americans with end-stage renal disease are kept alive by dialysis or renal transplantation at a cost of over $10 billion per year yet no occupational disease appears in the list of causes of renal failure in the comprehensive annual U.S. Renal Data System report. The failure to recognizeApril 11, 2000 occupational renal diseases can be attributed in part to the difficulty in assigning etiology following a long latent period, the small number of workers who develop chronic renal failure, the difficulty in identifying interstitial nephritis because of the absence of heavy albuminuria, the non-specific histologic appearance of interstitial nephritis when renal failure is advanced, and by the concentration of medical resources on treatment rather than prevention.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Satoshi Ishikawa, M.D.** Saturday, 9:30 a.m., June 10, 2000

SPEECH TITLE: **Chemical Sensitivity Patients in Japan, Part I**

The information below has been provided by the speaker.

1.) **Goals and objectives:** Establishment of diagnosis in patients with chemical sensitivity y neuro-ophthalmological technique.

2.) **Outline of talk/abstract:** Our results of neuro-opthalmological approach such as pupillography, eye movement (smooth pursuits) and contrast sensitivity to the patients with chemical sensitivity will be introduced.

3.) **Conclusion of what is to be learned:** Diagnosis by neur-opthalmology technique is accurate, inexpensive and non-invasive. The significance of those tests will be emphasized.

4.) **References:**

1. Ishikawa S et al. Evaluation of the autonomic nervous system response by pupillographical study in the chemical sensitive patient.
2. Ishikawa et al. Comparison of threshold visual Env Med. 8(4)121-127, 1991. Perimetry and objective pupil perimetry.
3. J. Neuro-ophthalmology 19(2)89-99,1999

Notes:

CHEMICAL SENSITIVITY PATIENTS IN JAPAN (PART I)

Satoshi Ishikawa, M.D.

Professor of Emeritus, Kitasato University, Tokyo, Japan

Ishikawa, one of the author, reported neurotoxicity of organophosphate pesticides even domestic use as early as in 1970. When epidemiological study was made that time at Saku area where helicopter spray of malathion was made, inhabitants of the sprayed area (children of Saku, N=49 with the mean age of 9.0 years) complained of headache, nausea and vomiting, stomach pain, numbness of the lower legs with fatigue, photphobia and narrowing of the lids. These complaints were significantly higher that the controls (children in Tokyo, N=49 with the mean age of 9.8 years). Those results were published from Neuro-toxicity of the Visual System Raven Press 1980 ed Merigan et al. Almost the same complaints were seen in the patients with a possible chemical sensitivity patients who had and environmental contact with anti-termite (such as fenitrothion and chlorpyrifos) and plasticizers, flame retardants and lubricants. In order to diagnose the patients with a possible chemical sensitivity, we have constructed ECU (Environ-mental Control Unit) with the help of Mitsubishi-related Company of Shinryou-Reinetsu with in part a technical advice of Dr. William Rea. In this presentation, we would like to introduce our Kitasato-ECU together with the results of the double blind study of low-dosage of gases of formaldehyde and toluene.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Cyril W. Smith Ph.D.** Friday, 1:30 p.m., June 9, 2000

SPEECH TITLE: **EM Fields in Health, in Therapies, as Hazards**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To provide insight into the good and bad effects of the EM environment on living systems.

2.) **Outline of talk/abstract:** The normal involvement of EM fields and frequencies in healthy living systems and the interactions with water, leads to their discussion in relation to acupuncture, homoeopathy, serial dilutions of allergens and environmental toxins, and to ways in which they might become hazardous.

3.) **Conclusion of what is to be learned:** The integration of your clinical and biochemical background with the bioelectromagnetics of water and living systems.

4.) **References:**

1. Smith CW. In: Taddei-Ferretti C and Marotta P (Eds.) High Dilution Effects on Cells and Integrated Systems, Series on Biophysics and Biocybernetics Vol 3 - Biophysics. Singapore: World Scientific, 1998, ISBN 981-02-3216-0.
2. *Coherence in biological systems and water.* pp.88-94.
3. *Water and the diagnosis and treatment of electromagnetic hypersensitivity.* pp.184-192.
4. *Water and bio-communication.* pp.295-304.
5. Smith C.W. Is a living system a macroscopic quantum system? *Frontier Perspectives*, 7(1), 9-15 (1998), (Temple University, Philadelphia, audio tape of 1997 lecture from Frontier Sciences Department). ISSN:1062-4767.
6. Smith CW. Physicks and Physics. *The J. of Alternative and Complementary Medicine* **5(2)**: 191-193, April 1999.

Notes:

EM FIELDS IN HEALTH, IN THERAPIES, AS HAZARDS

Cyril W. Smith Ph.D.

School of Acoustics and Electronics, University of Salford

An electromagnetic environment is essential to the well-being of all living systems. Beyond the thermal and photosynthetic effects of sunlight, there is a normal involvement of environmental fields and frequencies with healthy living systems and their endogenous frequencies. Water has remarkable properties in respect of coherent frequencies extending far beyond chemical bond lengths and a "memory" capability for frequencies which will be discussed. There is a fundamental duality between frequency and the chemical bond, without which chemical analysis by spectroscopy would be impossible. Within the range of coherence, there may be many frequencies with proportional velocities at which coherence propagates, but all with the same wavelength. This allows interactions between widely different parts of the spectrum.

There are endogenous frequencies associated with acupuncture meridians and their target organs which can be entrained by environmental frequencies, by chemical frequency signatures, by homeopathic potencies and by serial dilutions of allergens and environmental toxins. If entrainment tends to move the living system towards what is it normality, the frequencies are therapeutic. In normality, frequencies fluctuate slightly according the endogenous electric activity. In any closed-loop feedback regulatory system, cause-and-effect cannot be distinguished. Frequencies might become hazardous if through chronic exposure a living system adapts into a frequency pattern corresponding to some disease state. Frequency patterns can be imprinted into water and living systems and they will transmit onwards to cultured daughter cells.

ABSTRACT INFORMATION & NOTES

Speakers Name: **William Rea, MD** Saturday, 11:00 a.m., June 10, 2000

SPEECH TITLE: **The Environmental Aspects of Cardiovascular Disease, Part II**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To better understand the environmental aspects of cardiovascular disease.

2.) **Outline of talk/abstract:** To learn how to use in the office the knowledge acquired to enhance the patient's treatment.

3.) **Conclusion of what is to be learned:** The outcome of these problems is very successful when causes are found and eliminated.

4.) **References:**

Notes:

THE ENVIRONMENTAL ASPECTS OF CARDIOVASCULAR DISEASE, PART II

William J. Rea, MD, FACS, FAAEM

Environmental Health Center - Dallas

Patient with small vessel either lymphocytic or leukocytoclastic vasculitis is the most common vascular abnormalities seen in environmental medicine. This series of patients seen at the Environmental Health Center - Dallas will be discussed. The series runs into the thousands but here 100 cases are viewed and defined and causes, triggering agents, autonomic nervous system changes, immune and toxic parameters and management results are discussed.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Theodore R. Simon, MD** Saturday, 11:30 a.m., June 10, 2000

SPEECH TITLE: **Nuclear Cardiology 2000**

The information below has been provided by the speaker.

1.) **Goals and objectives:** Nuclear cardiology can answer many common clinical questions that confront health care professionals. This presentation seeks to address these questions and their answers.

2.) **Outline of talk/abstract:** The presentation develops appropriate clinical questions and discusses the general advantages of the Nuclear Cardiological approach. These questions begin with common cardiac diseases, such as coronary artery disease and both intracardiac and extracardiac shunts. They then extend from diagnosis to clinical management of both surgical and medical therapies. Additional questions relate to concurrent processes, such as diabetes mellitus. Questions expand into evaluation of cardiotoxicity from chemotherapy and other drugs.

Having established the astute use of this armamentarium, the topics focus on the examinations to help the health care provider develop proper expectations for nuclear cardiology laboratory support including analytic techniques as well as both equipment and radiochemistry.

3.) **Conclusion of what is to be learned:** Nuclear Cardiology offers answers to common questions presented to the astute clinician using methodologies that are safe, and relatively easy to tolerate.

4.) **References:** Abbreviated References:

1. Bax *et al. J Nucl Med* 1998;39:1481-1486
2. Franken *et al. J Nucl Med* 1996;37:718-722
3. Matsuo *et al. J Nucl Med* 1996;39:712-717
4. Merlet *et al. J Nucl Med* 1883;33:471-477
5. Merlet *et al. J Nucl Med* 1999;40:224-231
6. Merlet *et al. J Nucl Med* 1999;40:917-923
7. Otsuku *et al. J Nucl Med* 1997;38:567-572
8. Sciagra *et al. J Nucl Med* 1999;40:363-370
9. Vallabhajosula *et al. J Nucl Med* 1997;38:1788-1796
10. Wakasugi *et al. J Nucl Med* 1992;33:208-214
11. Weissman *et al. J Nucl Med* 1996;199:353-357

Notes:

ABSTRACT INFORMATION & NOTES

Speakers Name: **Thomas Croley, Ph.D.** Saturday, 1:30 p.m., June 10, 2000

SPEECH TITLE: **Effective use of Cold Laser Therapy in the Treatment of Environmentally Challenged Patients**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To better understand and learn about cold laser therapy.

2.) **Outline of talk/abstract:**

A.) What is cold laser?

B.) Why is cold laser therapy effective?

C.) What conditions have been treated successfully with cold laser?

D.) What is the specific mechanism of action of cold laser on tissues?

E.) How has cold laser therapy helped the environmentally challenged patients?

3.) **Conclusion of what is to be learned:**

4.) **References:**

Notes:

ABSTRACT INFORMATION & NOTES

Speakers Name: **William J. Meggs, MD, Ph.D.** Saturday, 2:00 p.m., June 10, 2000

SPEECH TITLE: **Allergy and the Heart**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To understand that the heart can be a primary target organ of allergic reactions.

- To know the anatomy and physiology of allergy reactions involving the heart.
- To gain a familiarity with clinical and research studies that verify the primary effects of allergic reactions on the heart

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:** Allergic reactions can have direct adverse effects on the heart that lead to morbidity. Adverse effects include cardiac arrhythmias, ischemia, myocarditis, coronary vasculitis, and myocardial infarction. Allergic reactions should be considered in the differential diagnosis of any patient with cardiac disease.

4.) **References:**

1. Carloss H. transient electrocardiograhi changes in anaphylaxis. Southern Medical journal 1976;69:1621-2
2. Criep LH, Woehler TR. The heart in human anaphylaxis. Annals of allergy 1971;29:399-409.
3. Frigen GA, Prager DJ. Experimental Cardiac anaphylaxis: physiologic, pharmacologic and biochemical aspects of immune reactions in the isolated heart. American Journal of Cardiology 1969;34:474-491.
4. Marone G, et al. Immunlogical characterization and functional importance of human heart mast cells. Immunopharmacology 1995;31:1-18.
5. Rea WJ. Environmentally triggered cardiac disease. Annals of Allergy 1978;40:243-251

Notes:

ALLERGY AND THE HEART

William J. Meggs, MD, Ph.D., FACEP

Professor and Vice Chair for Clinical Affairs, Department of Emergency Medicine,

Brody School of Medicine

The heart contains mast cells which release histamine and other allergic mediators when degranulated by antigens to which a patient is allergic. These mediators can have a direct effect on the heart. Research studies show that histamine exposure in isolated heart preparations produces tachyarrhythmias and decreased cardiac blood flow. Human studies demonstrate that similar findings in patients challenged with substances to which they are allergic. Allergic reactions to bee stings produce arrhythmias, electro cardiographic abnormalities, and myocardial infarction, in the absence of pre-existing coronary disease and treatment with drugs with cardiac effects. A number of drugs have been documented to produce allergic myocarditis. Cardiac allergy is seldom considered in the differential diagnosis of patients with heart disease which is often misdiagnosed as idiopathic.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Allan D. Lieberman, MD** Saturday, 2:30 p.m., June 10, 2000

SPEECH TITLE: **Pesticide Poisoning - The Missed Diagnosis**

The information below has been provided by the speaker.

1.) **Goals and objectives:** Impress the importance of the history of Environmental Exposures and recognize the manifestations of organophosphate poisoning.

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:** 1) Importance of exposure history, 2) Recognizing the many faces of pesticide poisoning.

4.) **References:**

1. Jamal, GA. Neurological Syndromes of Organophosphorus Compounds. Adverse Drug Reactions. Toxical Rev. 1997, 16(3), 133-170.
2. Devinsky, O., Kernan, J., Bear, D.M., J. Neuropsychiatry. 4:189-94, 1992.

Notes:

PESTICIDE POISONING - THE MISSED DIAGNOSIS

Allan D. Lieberman, MD

Center for Occupational & Environmental Medicine, P.A., N. Charleston, SC

Many patients we see often tell us that they have been seen by other physicians who simply can't understand what's wrong with them or suggest psychiatric consultation.

My experience as an occupational and environmental physician is that most physicians do not recognize the manifestations of Neurotoxicity and thus fail to make the correct etiologic diagnosis. The challenge, as well as fun of doing what we do is trying to identify causation for the myriad of signs and symptoms we see in our multi-system affected patients.

The case report becomes one of the most important obligations of our kind of practice. Only when multiple cases are seen to be associated with a specific environmental trigger, can a hazardous material be established as a cause of an enigmatic or bizarre disorder.

Today's report of a 31 year old woman is a case in point. Almost 30 years ago, the late Richard Macarness of England, gave a lecture to the Society For Clinical Ecology as the academy was once called. Mac was a psychiatrist, and is credited with being the father of Environmental Medicine in the United Kingdom. He quoted Sigmund Freud as saying "Someday there will be a physiological explanation for psychosis" and then Mac went on to say "and today we have that explanation. We call it clinical ecology".

How exciting that we in environmental medicine are in the right place at the right time and that we can recognize environmental triggers as cause of so many of our mental and behaviorally disordered patients.

Our patient said "31 years later, my life of strange illnesses is just now making sense. I've been like Dr. Jeckle and Mr. Hyde. No doctor could tell my why one minute I appear crazy and the next I'm fine."

A careful chronological history in our case demonstrated a clear temporal relationship between exposure to organophosphate pesticides and altered behavior. Whenever exposed, she became psychotic and symptomatic only to improve when removed from the exposure.

In establishing causation, the most important criterion is demonstrating a temporal relationship. However, it is also important to document that the incriminated chemical is capable of producing this effect. But what is no one else ever published these effects? Absence of previous toxic effect does not rule out toxicity, but emphasizes the importance of case reports. The scientific literature however, does describe chronic organophosphate induced psychiatric disorder (COPIND)1,2 Which manifests:

- Neurobehavioral changes
- Cognitive changes
- Mental manifestations
- Chronic fatigue
- Peripheral neuropathy
- Neuromuscular dysfunction

Our patient had all of these changes thus fulfilling this criterion for establishing causation as well as the following other criteria:

- The exposures and effect were documented
- The effects of injury are still present
- The dose was sufficient to produce injury
- There are no other reasonable diagnoses to explain the disease or disorder

In a world heavily contaminated by pesticides and especially organophosphate pesticides, I feel strongly that every physician should be aware of what these chemicals can do.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Jon B. Pangborn, Ph.D.** Saturday, 3:30 p.m., June 10, 2000

SPEECH TITLE: **Update on Clinical Aspects of Homocysteine**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To review the physiological implications of elevated homocysteine, associated clinical aspects, and methods of clinical assessment.

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:** Regardless of analytical procedure, a finding of elevated homocystine, homocysteine, homocyst(e)ine or total homocysteine is cause for concern, not only for susceptibility to occlusive arterial disease but for oxidant stress, atherosclerosis, as well as neuropsychiatric problems. Assessment by quantitative amino acid analysis is most informative as to cause. The occurrence of elevated homocysteine, including that provoked by methionine challenge, is one in 70 (about 4,000,000 of the US population).

4.) **References:**

1. Bremer H.J. et al. Disturbances of Amino Acid Metabolism: Clinical Chemistry and Diagnosis, Urban & Scwarzenburg, 93-101 (1981).
2. Mudd S.H. and H.L. Levy "Disorders of Transsulfuration:, Chapt. 25 in Stanbury et al., eds, The Metabolic Basis of Inherited Disease, 5th ed. (1983).
3. Boers G.H.J. et al. "Heterozygosity for Homocystinuria in Premature Peripheral and Cerebral Occlusive Arterial Disease" N.E.J. Med. Vol.313 no.12, 709-715 (1985).
4. Lindenbaum J. et al. "Neuropsychiatric Disorders Caused by Coblamin Deficiency in the Absence of Anemia or Macrocytosis" N.E.J.Med. Vol.318 no 26, 1720-1728 (1988).
5. Stampfer M.J. et al. "A Prospective Study of Plasma Homocyst(e)ine and Risk of Myocardial Infarction in US Physicians" JAMA vol.268 no.7, 877-881 (1992).
6. Wu J.T. et al. "Conversion of a Qualitative Screening Test to a Quantitative Measurement of Urinary Cystine and Homocystine" Ann. Clin. & Lab.Sci. Vol..22 no.1, 18-29 (1992).
7. Ueland P.M. et al. "Total Homocysteine in Plasma or Serum: Methods and Clinical Applications" Clin.Chem. Vol.39 no.9, 1764-1779 (1993).
8. Selhub J. et al. "Association Between Plasma Homocysteine Concentrations and Extracranial Carotid-Artery Stenosis" N.E.J.Med. Vol.332 no.5, 286-291 (1995).
9. Ueland P.M. "Homeocysteine Species as Components of Plasma Redox Thiol Status", Clin.Chem. Vol.41 no.3, 340-342 (1995).
10. Stamler J.S. and A. Slivka "Biological Chemistry of Thiols in the Vasculature and in Vascular-related Disease" Nutr. Revs. Vol.54 no.1, 1-30 (1996).
11. Loscalzo J. "The Oxidant Stress of Hyperhomocyst(e)inemia" J.Clin. Invest. Vol.98 no.1, 5-7 (1996).
12. Bellamy M.F. et al. "Hyperhomocysteinemia After an Oral Methionine Lad Acutely Impairs Endothelial Function in Healthy Adults" Circulation vol.98 1848-1852 (1998).

UPDATE ON CLINICAL ASPECTS OF HOMOCYSTEINE

Jon B. Pangborn, Ph.D.

A normal intermediary metabolite of the essential amino acid methionine, homocysteine can become elevated in cells and body fluids die to one or more of several circumstances-

- Deficiency or dysfunction of vitamin B6 or pyridoxal 5-phosphate, the coenzyme for cystathionine beta-synthase (the enzyme that catalyzes formation of cystathionine from homocysteine and serine)
- Deficiency of serine (rarely observed)
- Deficiency of vitamin B12 or of its cofactor form, methyl-cobalamin, which methylates homocysteine to re-form methionine
- Deficiency of folic acid or of its cofactor form, 5-methyl-cobalamin, which methylates homocysteine to re-form methionine
- Genetic weakness in the cystathionine beta-synthase enzyme (possibly countered with vitamin therapy)
- Genetic weakness in the folate-B12 methyl transfer enzymes (5, 10-methylene tetrahydrofolate reductase, E.C.1.1.99.15 or 5-methyltetrahydrofolate-homocysteine methyltransferase, E.C.2.1.1.13).

A host of physiological and pathological disorders have been traditionally associated with above-listed nutritional and metabolic deficiencies or dysfunctions. Mudd, et al., lists over 30 for just cystathionine -synthase weakness including ocular problems (mytopia, ectopia lentis), skeletal conditions (osteoporosis, scoliosis), CNS disorders/neurological problems (seizures, mental retardation, psychiatric disorders) vascular disease (acclusive), and others (myopathy, brittle hair, fatty liver, inguinal hernia).

Elevated homocysteine is now understood to provoke oxidant stress leading perhaps to oxidation of membrane lipids and cholesterol, thus increasing risk for CVD. Homocysteine in blood readily forms disulfides (homocystine, homocysteine-cysteine, homocysteine-thiols) with concurrent production of superoxide ion, O2. The resulting hydrogen peroxide and hydroxyl radical activity makes homocysteine a cause of endothelial tissue damage and a possible cause of atherosclerosis.

Recent studies have also connected elevated homocysteine to mental depression and Alzheimer's disease; however, causal mechanisms are uncertain. It is possible, for example, that disordered methylation affects catecholamine metabolism, neurotransmitter chemistry and homocysteine levels, and that homocysteine is symptomatic rather than causal.

Assessment of homocyst(e)ine (homocysteine + homocystine) status is done by amino acid analysis of blood plasma or urine. What is actually measured (and the associated reference ranges) depends upon analytical procedures. Urine amino acid analysis by HPLC usually measures only the oxidized, disulfide form, homocystine. Plasma HPLC analysis may measure only homocysteine. Non-HPLC usually measures only the oxidized, disulfide form, homocystine. Plasma HPLC analysis may measure only

homocysteine. Non-HPLC "Screening" tests may measure blood plasma or serum homocysteine and various disulfides including homocystine, homocysteine-cysteine and homocysteine thioethers. "Total homocysteine" (in blood) includes these species plus protein-bound homocysteine. Although quantitative amino acid analysis only measures homocysteine and/or homocystine, it is most informative about causation because it also measures methionine, serine, cystathionine and other analytes sensitive to vitamins B6, B12, and folate.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Malcolm Beck** Saturday, 4:00 p.m., June 10, 2000

SPEECH TITLE: **The Secret Life of Compost**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To show that all organics must be recycled

2.) **Outline of talk/abstract:** Explain how nature works, necessity of energy in the soil, structure organic give soil, air, water quality & quantity, and food depends on soil quality

3.) **Conclusion of what is to be learned:** The soil determines the quality of all life on earth, let the soil degrade and all life on earth degrades to the same degree

4.) **References:**

A life time farming/growing organic, many, many books, papers, lectures, my own research.

Notes:

THE SECRET LIFE OF COMPOST
LIFE, DEATH, DECAY AND MANS EXISTENCE

Malcolm Beck

Walk into the woods and meadows and visit with Nature. You will be in the presence of much life. Many types of plants, insects and animals - large and small. There will be life in abundance. Now take a closer look. There is an equal amount of death. There will be

dead grass and leaves, fallen limbs and trees, even dead animals and insects, all laying as a mulch, composting the earth.

When a plant or animal dies, even though it may be consumed higher in the food chain, it will eventually be eaten by the decomposing microbes. They will decay or disassemble it and put it back into the soil. If they didn't, our planet would now be miles deep in dead things.This life-death-decay cycle has built the thin layer of fertile soil that covers our land. It nourishes and grows the many plants which bridge the life from the soil to man.

In the beginning, our planet was just a round mass of mineral moving in it's planned orbit through space. At some point, the Almighty saw fit to breath life into earth, very meager and primitive life, but life with a crucial mission.

As these micro-forms of life lived and reproduce, they fed on and etched away at the rocky mineral earth surface. Their exudes and rotting remains formed humus and mild acids to etch away still more minerals. This process went on and on until small amounts of our first soil was formed.

Since then, the life, death, and decay of each preceding life form has been creating better conditions for future life forms than were there before. The decay process builds with added interest to the soil's bank account, and after countless centuries of creating conditions for higher and more complex forms of life, Man, the most complex of all life, was able to exist and be sustained.

Man has accumulated much knowledge, but in areas of his healthy existence he seems to be slow to learn. Man sees death as a loss, or something to be sorrowful of, and he considers decay as something ugly. He doesn't understand why Nature always returns the dead back to the soil from where it came.

In Nature, there is no waste. All is reused, and usually mad into something of still greater value for the sustenance of life.

If man truly understood the laws of recycle and return, he would without delay return all organic waste back to the land from where it came.

If man continues to break this law of return, he will not only stop the life generating processes of the soil. He will cause the soil to degenerate - a process that will sooner or later degrade all life ... including man himself.

Sunday Morning Session

8:15 **ANNOUNCEMENTS/MODERATOR: David Hickey, M.D.**

8:30 "Cell Cycles and Nutrition", Bertie Griffiths, Ph.D.

8:50 Q & A

9:00 "Chemical Sensitivity patients in Japan, Part II", Satoshi Ishikawa, M.D.

9:20 Q & A

9:30 "Endocrine Disruptors in the Bitterroots", Doug Seba, Ph.D.

9:50 Q & A

10:00 BREAK WITH EXHIBITORS

MODERATOR: Cyril Smith, Ph.D.

10:30 "Toxins and the Heart", William Meggs, M.D.

10:50 Q & A

11:00 "Heart Rate Variation in Adults Exposed to Chlorine and Creosol", Kaye Kilburn, M. D.

11:20 Q & A

11:30 "Electromagnetic Screening as a Protection From Telephone Frequencies and Effects on Heart Rate Variability.", Jean Monro, M.D.

11:50 Q & A

12:00 SUMMARY AND CLOSE: William J. Rea, M.D.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Bertie Griffiths, Ph.D.** Sunday, 8:30 a.m., June 11, 2000

SPEECH TITLE: **Response of the Cell Cycle to Nutrition**

The information below has been provided by the speaker.

1.) **Goals and objectives:**

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:**

4.) **References:**

Notes:

RESPONSE OF THE CELL CYCLE TO NUTRITION

Bertie Griffiths, Ph.D.

Environmental Health Center - Dallas

ABSTRACT INFORMATION & NOTES

Speakers Name: **Satoshi Ishikawa, M.D.** Sunday, 9:00 a.m., June 11, 2000

SPEECH TITLE: **Chemical Sensitivity Patients in Japan, Part II**

The information below has been provided by the speaker.

1.) **Goals and objectives:** Establishment of diagnosis in patients with chemical sensitivity y neuro-ophthalmological technique.

2.) **Outline of talk/abstract:** Our results of neuro-opthalmological approach such as pupillography, eye movement (smooth pursuits) and contrast sensitivity to the patients with chemical sensitivity will be introduced.

3.) **Conclusion of what is to be learned:** Diagnosis by neur-opthalmology technique is accurate, inexpensive and non-invasive. The significance of those tests will be emphasized.

4.) **References:**

1. Ishikawa S et al. Evaluation of the autonomic nervous system response by pupillographical study in the chemical sensitive patient.
2. Ishikawa et al. Comparison of threshold visual Env Med. 8(4)121-127, 1991. Perimetry and objective pupil perimetry.
3. J. Neuro-ophthalmology 19(2)89-99,1999

Notes:

CHEMICAL SENSITIVITY PATIENTS IN JAPAN (PART II)

Satoshi Ishikawa, M.D.

Professor of Emeritus, Kitasato University, Tokyo, Japan

Indoor air chemical pollution and human health have been studied. During pas 10 years, we saw the Patients with chemical Sensitivity (CS) at the Department of Neuro-opthalmology Clinic at Kitasato University Hospital. Since we considered that we need to obtain further knowledge of this syndrome. The patients had indefinite complaints and most of the physicians in Japan dismissed the patients as psychogenic disorders. However, there is a difference between CS and the patients with psychogenic disorders. We have applied a technique of pupil analysis for quantitative study of CS patients and published a paper in Environmental Medicine 8:121-127, 1991. The diagnostic criteria of MCS was made in 1997 with the aid of The Japanese Ministry of Health and Welfare. Based on this criteria, we collected 141 cases and carried out the following neuro-ophthalmologic tests. A routine blood and urine, blood chemistry, immunology and ECG examinations did not help for the diagnosis. However, neuro-opthalmological examinations did not help for the diagnosis. However, neuro-ophthalmological examinations helped to quantitate the patients grade of impairment especially at the central nervous system. The tests carried out were (1) contrast sensitivity, (2) pupil response to the light, (3) Eye movement especially smooth pursuit and the limited cases underwent to, (4) Challenge test using low dosage aspiration of formaldehyde, toluene, xylene, and methane under blind manner. The results indicated that the chemicals of formaldehyde, toluene, phenol, xylene, VOCs and ant-termite such as organophosphorus compounds are the major chemicals to produce CS.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Doug Seba, Ph.D.** Sunday, 9:30 a.m., June 11, 2000

SPEECH TITLE: **Endocrine Disruptors in the Bitterroots**

The information below has been provided by the speaker.

1.) **Goals and objectives:**

- To review the apparent toxic effects of humans and animals from fungicides used on potato crops in Idaho.
- To review the current knowledge for multi-system fungicidal toxicity to humans and animals in Montana.
- To understand how to review and rebut critiques of this work as a classical role model for Environmental Medicine research in general.

- To present some treatment modalities and speculate on homeopathic healing of exposed wildlife.

2.) **Outline of talk/abstract:** Review latest findings and present graphic display of effects.

3.) **Conclusion of what is to be learned:** That a series of disparate events came together to reconcentrate endocrine disruptors to levels that, while low in absolute environmental terms, are sufficiently high to cause widespread harm in wildlife and possibly humans.

4.) **References:**

- "Potential Effects of Pesticides on Deer and Other Wildlife in The Bitterroot Valley, MT." by Anne Fairbrother. A report prepared for the Lee Metcalf National Wildlife Refuge, U.S. Fish and Wildlife Service, by Parametrix, Inc. 1600 SW Western Blvd, Suite 165, Corvallis, OR 97333. 34p. January 25, 2000.
- "Wildlife and Endocrine Disrupters: Requirements For Hazard Identification." by Pamela M. Campbell and Thomas H. Hutchinson. Environmental Toxicology and Chemistry 17:127-135, 1998.
- The Presence of Organochlorine Pesticides and Chlorinated Hydrocarbon Solvents in the Blood Chemically Sensitive Patients. A Statistical Comparison with Therapeutic Medication and Other Highly Potent Natural Hormones." by William J. Rea, Ervin J. Fenyves, Douglas Seba, and Yaquin Pan. Journal of Nutritional & Environmental Medicine. In press.
- "Pesticide Appliers, Biocides, and Birth Defects in Rural Minnesota." by Vincent F. Garry. Environmental Health Perspectives 104:394-399, 1996
- "Evidence of Currently-Used Pesticides in Air, Ice, Fog, Seawater and Surface Microlayer in the Bering and Chukchi Seas." by Sergey M. Chernyak, Clifford P. Rice and Laura L. McConnel. Marine Pollution Bulletin 32:410-419, 1996
- "Ambient Air Concentrations of Pesticides in California." by Lyton W. Baker et. al. Environmental Science and Technology 30:1365-1368, 1996.
- Global Distribution of Persistent Organochlorine Compounds." by Staci L. Simonich and Ronald A. Hites. Science 269:1851-1855, 1995.
- Atmospheric Pollutants and Trace Gases: Vapor-Phase and Particulate-Associated Pesticides and PCB Concentrations in Eastern North Dakota Air Samples." by Steven B. Hawthorne et. al. Journal of Environmental Quality 25:594-600, 1996
- "Pregnancy Outcome Following Gestational Exposure to Organic Solvents." by Sohail Khattak et. al. Journal American Medical Association 281:1106-1109, 1999
- "Chemicals Implicated in Amphibian Decline." by Bette Hileman. Chemical & Engineering News 77:22-23, 1999
- "Tall Mountains Collect More Pollutants." by Elizabeth Wilson. Chemical & Engineering News 76:16, 1998
- "Fungicide, Factsheet: Chlorothalonil," by Caroline Cox. Journal of Pesticide Reform 17:14-20, 1997
- "Innovative Cropping Systems Can Replace Hazardous Pesticides." by Karen Murphy. Journal of Pesticide Reform 17:2-6, 1997

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Notes:

ENDOCRINE DISRUPTORS IN THE BITTERROOTS

Douglas B. Seba and Judy Hoy*

Independent Marine Scientist, Key West, FL

First, presented in preliminary form at this conference last year, this paper will expand upon an active year of research into the harmful effects of endocrine disruptors on broad spectrum of animals undergoing rehabilitation at a facility in the remote Bitterroot mountains of Montana.

After 25 years of successful wildlife rehabilitation, the second author began noticing anomalies in the sex organs of large mammals, particularly deer, in 1994. By 1997 it was clear that this was not individual variation but a trend not seen previously. By 1999, these variations had spread to a variety of wildlife along with alterations not related to sex organs. Human health effects were also noted.

These trends developed at the same time that a potato blight began in the fields of Idaho, a blight which worsened in 1997, and continues to present. Concurrently, a great deal of fungicides were applied in a continuing attempt to control the blight, particularly Chlorothalonil and Dithane.

Prevailing winds blow up-slope from Idaho potato fields into the Bitterroot mountains of Montana before they cross the continental divide. Atmospheric research has shown that, surprisingly, mountains collect considerably more environmentally persistent organochlorine compounds than flatlands. When winds blow up-slope, they condense with the lowering of air temperature and fractionate dramatically. A difference of only a couple of thousand meters can result in concentration gradients of 10 or 100-fold. Late season blight fungus spores, which are much larger and heavier than the fungicides used on them are routinely carried hundred of miles on moist weather fronts into the Bitterroot mountains and the presence of Chlorothanlonil has been documented at the remote wildlife rehabilitation center.

During the last year extensive pathology has been conducted on large animals in the Bitterroot mountains showing a wide-spread problem with calcium metabolism at many levels which is classic mark of environmentally induced endocrine disruptors. This pathology will be reviewed along with other on-going research.

Treatment with homeopathic cell salts and electrolytes has led to some dramatic healings in wildlife and these techniques will also be reviewed.

Finally, this research has prompted an investigation by Federal wildlife authorities through a contractor to issue a report dismissing the entire issue as a non-event. This report will be examined in considerable detail as it is a classical example of setting up a strawman to draw attention away from a disturbing trend. This can be used as a role model for many research reports from the field of Environmental Medicine.

The authors wish to thank the Academy of Marine Sciences in Ft. Lauderdale for its continuing support of this research.

*Complete sets of data and/or data exchange for this non-profit facility can be obtained from Ms. Judy Hoy, Bitterroot's Wildlife Rehabilitation Center, 2858 Pheasant Lane, Stevensville, MT 59870. E-mail: adcoon@bitterroot.net Phone 406/777-2487

ABSTRACT INFORMATION & NOTES

Speakers Name: **William J. Meggs, MD, Ph.D.** Sunday, 10:30 a.m., June 11, 2000

SPEECH TITLE: **Toxins and the Heart**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To be able to recognize the most commonly encountered cardiac toxins.

- To understand the cardiac effects of acute and chronic toxic exposures.
- To learn an approach to evaluating and treating patients with exposures to toxins that can have cardiac manifestations.

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:** The participant should know the cardiac effects of a number of toxins, and be able to diagnose and treat patients with these exposures.

4.) **References:**

1. Derlet RW. Horowitz BZ. Cardiotoxic drugs. Emergency Medicine Clinics of North America. 13(4):771-91,1995 Nov
2. Goldfrank's Toxicological Emergencies, 6th Edition. Goldfrank LR et al, editors. Appleton & Lange, Stamford, CT, 1998.
3. Kelly RA. Smith TW. Recognition and management of digitalis toxicity. [Review] [41 refs] American Journal of Cardiology. 69(18):108G-118G

4. Meggs WJ. Cardiovascular manifestations of systemic diseases. In: Aghababian RV, ed. *Emergency Manifestations of Cardiovascular Disease.* Boston, butterworth Heinemann; 1994:293-305.
5. Roth A. Zellinger I. Arad M. Atsmon J. Organophosphates and the heart. [Review] [53 refs] Chest. 103(2):576-82, 1993 Feb.

Notes:

TOXINS AND THE HEART

William J. Meggs, MD, Ph.D., FACEP

Professor and Vice Chair for Clinical Affairs, Department of Emergency Medicine,

Brody School of Medicine

A number os substances have cardiac manifestations that should be understood by all practicing physicians. Prescription drugs such as theophylline, tricyclic antidepressants, digoxin, beta blockers, and calcium channel blockers have cardiac toxicity from adverse reactions, chronic overdoses, and scute overdoses. The specific toxicities of a number of drugs and approaches to treatment will be reviewed. Organic solvents can be toxic to the heart, and cardiac toxicity is a common cause of death among glue sniffers and other solvent abusers. A number of metals are toxic to the heart, and thes4e will be reviewed. Drugs of abuse, most notably ethanol, cocaine, and amphetamines, can be toxic to the heart with chronic use and acute exposures, even in the absence of overdose. Cardiac toxicity is a manifestation of organophosphate poisoning. A number of common plants can also poison the heart if ingested.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Kaye H. Kilburn, M.D.** Sunday, 11:00 a.m., June 11, 2000

SPEECH TITLE: **Heart Rate Variation in Adults Exposed to Chlorine and Creosol**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To describe a simple recording and interpretive test for reduction or loss of respiratory sinus arrhythmia that distinguishes groups of people exposed to some chemicals.

2.) **Outline of talk/abstract:**

3.) **Conclusion of what is to be learned:** How to record and interpret heart rate so as to distinguish respiratory variation. That reduction or loss of respiratory sinus arrhythmia occurs from some solvents and chlorine/creosol.

4.) **References:**

1. Matikainen E, Juntunen J: Examination of the peripheral autonomic nervous system in Occupational Neurology, Neuro behavioral methods in Occupational and Environment Health. World Health Organization
2. Copenhagen 1985.
3. Morrow LA, Steinhauer SR: Alterations in heart rate and papillary response in persons with organic solvent exposure. Biol Psychiatry 1995;37:721-730.
4. Murata K, et al.: Changes in autonomic function as determined by ECG-R-R interval variability in sandal, shoes and leather workers exposed to n-hexane, xylene and toluene. Neurotoxicology 1994;15:867-876.

Notes:

HEART RATE VARIATION IN ADULTS EXPOSED TO CHLORINE AND CREOSOL

Kaye H. Kilburn, M.D.

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Normally during the inspiratory phase of respiration, heart rate increases. This vagus nerve regulated sinus arrhythmia is reduced or abolished by aging, diabetes mellitus and solvent exposure. We determined whether an incident environmental exposure to chlorine-creosol of less than a day reduced RR variation.

We studied 58 adults subjects who had inhaled chlorine-creosol 3 years earlier and 22 adult subjects unexposed to chemicals. A standard electrocardiograph (ECG) machine was interfaced to a 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 ± 10% mean time. The percentage of beats outside the confidence band were compared.

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 and significantly different ($p < 0.043$). Thus in exposed groups sinus arrhythmia was reduced.

Exposure to chlorine/creosol reduced the respiratory variation of the heart rate. This effect on vagal function paralleled other adverse effects on the brain's regulation of balance, of hearing and choice reaction time, color discrimination, contrast sensitivity and visual field performance. The ease of this type of vagal testing and the linkage of vagal control of bodily function to the brains hippocampus, to emotion and to recall and

memory make this approach likely to help understand how chemicals alter automatic and volitional functions.

ABSTRACT INFORMATION & NOTES

Speakers Name: **Jean Monro, MD** Sunday, 11:30 a.m., June 11, 2000

SPEECH TITLE: **Electromagnetic Screening as a Protection from Telephone Frequencies and Effects on Heart Rate Variability**

The information below has been provided by the speaker.

1.) **Goals and objectives:** To illustrate the dangers of electromagnetic fields.

2.) **Outline of talk/abstract:** The effects of EM fields on screening using Snow Shield will be used with heart rate variability assessments.

3.) **Conclusion of what is to be learned:** The benefits of screening.

4.) **References:** Sait JL, Wood AW, Sadafi HA, A study of heart rate and heart rate variability in human subjects exposed to occupational levels of 50 Hz circularly polarized magnetic fields; Med Eng Phys 1999 Jun;21(5): 361-9.

Notes:

ELECTROMAGNETIC SCREENING AS A PROTECTION FROM TELEPHONE FREQUENCIES AND EFFECTS ON HEART RATE VARIABILITY

Jean Monro, MD

Medical Director of Breakspear Hospital, England

GOALS AND OBJECTIVES: In the first presentation heart rate variability will be shown before, during and after neutralization testing. The second presentation will review and discuss heart rate variability with and without electromagnetic effects, to illustrate the dangers of electromagnetic fields.

BACKGROUND: One hundred years ago there were no domestic electrical devices. A century later, in the western world it is impossible to find people who do not live or work in an electrical "smog".

This electric pollution may cause a syndrome called "Electrical Sensitivity", and typical symptoms include headache, depression, muscular weakness, inco-ordination and even blackouts. These symptoms may be caused in electrically sensitive people by many types of electromagnetic radiation including radio and microwaves, infra-red, normal and ultra-violet light, x-rays, and cosmic radiation.

At present, there are few doctors and scientists who fully appreciate the fundamental importance of electromagnetic radiation as the cause of a wide variety of illnesses. More importantly, so few doctors understand that electromagnetic radiation may offer us a new way of curing many diseases and symptoms, previously unresponsive to treatments with present methods. Up until the present the principles of physics have been used almost solely for diagnostic rather than curative purposes. X-rays, ultrasound scans, nuclear magnetic resonance and CAT scans are invaluable in the diagnosis of disease, however, it is predicted that in the 21st century electromagnetic radiation will be used for its untapped potential as a curative gent.

As a general rule the most dangerous devices are high current devices and those that require a very strong magnetic field for their operations like motors and transformers. Electrical sensitivity affects at least 1 in 1,000 of the population. Almost all electrically sensitive people are also sufferers from food and/or chemical allergies.

Non-ionising electromagnetic radiation is able to cause a wide variety of symptoms especially those related to blood vessels and the brain and nerves. These symptoms include flushing, blushing, palpitations, diarrhea, muscular aches, pins and needles especially in the hands and feet, dizziness, fits and blackouts, disorientation, headaches, noises in the head, depression and suicide, and persistent tiredness unrelieved by rest. Electrical sensitivity may also mimic neurological diseases such as paralysis, epilepsy and multiple sclerosis. Other diseases and symptoms associated with prolonged exposure to magnetic and electric fields include headache, depression, suicide, miscarriage, cancer and leukemia.

The reasons that electricity is often poorly recognized as a cause of illness is because of the fact that there is often a long latent period, 3-5 years, between exposure to the current and the effect being noticed. In addition, there may be a further 10 years before a well defined disease shows itself.

The human body is highly electrically active. Minute currents can be measured from every cell in the body and individual organs such as the heart and brain are routinely monitored to assess disease. Electrical activity is absolutely fundamental to life and the state of health of the body can be tested electrically in several ways. One technique that was developed in Russia is Kirlian photography. In this technique a very high frequency electric current is applied to the body and that part photographed. The electrical discharge given off changes according to the state of health.

A technique has been to expose electrically sensitive patients to very weak non-ionising electromagnetic radiation. The frequency of the electromagnetic radiation can be very accurately controlled by using crystal controlled oscillators. This testing can be done double-blind, and in many hundreds of cases we found that an electrically sensitive patient may be ill using one frequency but can be healed by using a second frequency.

Our work at Breakspear Hospital has found that there is a very strong similarity between electrically sensitive patients' frequencies by using an electrical oscillator, and

duplicating the effect by giving patients provocation/neutralization testing. It is believed that the provocation/neutralizing testing does not work by rules of pharmacology or chemistry since the dilution of many solutions is so great that there should not be a single molecule of the original substance contained in the injection.

Another method of testing a patient's response to different frequencies is to use the technique of applied kinesiology.

Electrical fields can be screened by using an earthed Gaussian screen, also called a Faraday Cage. This screening may be carried out relative cheaply by papering the walls of rooms with ordinary aluminum foil which is then connected to earth.

A similar effect may be obtained by using a material which has been sprayed with silver which then conducts electricity. This material may even be made into clothes.

Unfortunately, it is very difficult to screen magnetic fields but individual devices can be greatly modified to reduce their electric and magnetic radiation. For example, we can advise you about low radiation computer monitors which give out one tenth of the level amount of radiation of standard devices.

By understanding the principles of biophysics it has been possible to treat a large number of patients sensitive to non-ionising radiation. We have seen patients who have become very ill when they have lived close to high voltage powerlines due to their exposure to 50 Hz. When they moved away from this powerful radiation their health improved and electrical sensitivity disappeared. We have seen people who have used personal radio transmitters such as mobile telephones and deaf aids transmitter who have become unable to work due to the radio transmitter making them feel tired all the time. We have had patients sensitive to marine and aeronautical radar frequencies who become ill when living near airports and coastal marine radar facilities.

Finally, we have had patients becoming ill due to the electrical oscillators found in their computers and quartz watches.

Treatment of all these cases has consisted of reducing the Total Load of allergic problem in air, food, water and also the Total Load of non-ionising electromagnetic radiation from all sources.

Provocation/neutralization injections can also be helpful to find the healing frequency for an individual patient and the patient may be given their frequency by holding a glass vial containing water energized at that frequency.

CONCLUSION: The relevance of pulse changes in people will be highlighted, and the benefits of electromagnetic screening will be outlined.