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Deciphering Depression

New Insights Into The Role Of Stress And Subtle Energies

Depression can be a manifestation of many different psychiatric and physical disorders, a normal response to certain types of stress, or result from various medications. Unless the cause can be clearly identified, depression usually represents a description, rather than a diagnosis. However, selecting optimal therapy or trying to provide an accurate prognosis may require a more specific and accurate categorization. Various attempts at classification

have been made based on etiology, symptomatology, or the clinical course. In that regard, Kraepelin observed in 1921 that many depressed patients followed a clinical course quite similar to others who had initially presented with an opposite or manic mood. He postulated that some forms of depression and mania were actually components of the same disorder, which he termed manic-depressive insanity, to distinguish it from the depression of *dementia praecox*, a diagnosis later replaced by schizophrenia. This classification was widely accepted for decades, until Leonhard and colleagues proposed a further subdivision into patients with only mania or depression (unipolar), and those with manifestations of both (bipolar), and the term manic-depressive insanity was subsequently replaced by bipolar disease to

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The Newsletter of
**THE AMERICAN INSTITUTE OF
 STRESS**

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minimize the confusion that existed in the field.

There have also been further attempts at subclassifications of bipolar disease. Nevertheless, these taxonomic efforts have not significantly improved our understanding of the cause, prognosis, or mechanism of action of various therapeutic options. As a consequence, other approaches have been pursued to satisfy these concerns. What is really required here, as in other areas of Psychiatry, are objective measurements that can be used for diagnosis, and to evaluate the efficacy of therapy.

A Neuroendocrine Disorder?

Bipolar disease appears to be associated with disturbances in the limbic system of the brain, and particularly the hypothalamus, which is the site of origin for most neuroendocrine activities and responses to stress. The hypothalamic-pituitary-adrenal cortex axis is primarily activated by stress, and elevated plasma cortisol and ACTH are also commonly observed in depressed patients. Cortisol levels do not show the normal suppression seen following the administration of corticosteroids in many of these individuals, and this forms the basis for the dexamethasone suppression test (DST) for

endogenous depression. Many manic and depressed patients also exhibit elevated thyroid hormone levels. This system can be tested by injecting thyrotropin-releasing hormone (TRH), which normally causes an exaggerated release of thyroid-stimulating hormone from the pituitary. A significant minority of patients with bipolar disease exhibit a diminished reaction, and since there is little overlap between the DST and TRH tests, these two abnormal hormonal responses do not appear to have a common basis. It is likely that other hypothalamic-pituitary-target organ axes may also be involved. Depression in women has long been linked to fluctuations in sex hormones just prior to menstruation or after giving birth. Its common association with menopause is reflected in what was commonly diagnosed decades ago as "involutional melancholia".

Subtle Energies And Neurotransmitters

Over the past few decades, interest has focused on abnormal brain neurotransmitter activities in depression. Attempts to correct these with monoamine oxidase inhibitors and tricyclic and tetracyclic drugs have been a mainstay of treatment. In addition to serotonin, dopamine and norepinephrine, alterations in melatonin secretion are also associated with depression, particularly in Seasonal Affective Disorder, (SAD). The significant benefits of light therapy in SAD and other types of depression, and anecdotal reports of the efficacy of cranioelectric stimulation, have increased interest in the therapeutic potential of such subtle energies. The beneficial effects of lithium may be due to its ability to decrease sensitivity to light.

All of the neuroleptic drugs act by simulating, stimulating, or antagonizing the effects of naturally produced neurotransmitters, and this is particularly true for antidepressant medications. However, in the final analysis, all these activities involve the exchange of weak electrical signals at specific receptor sites. While there are no distinctive diagnostic waking EEG patterns that will identify depressed patients, disruption of sleep is a common symptom,

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and sleep patterns have revealed consistent findings that differentiate depressed from normal patients, and even from those simply suffering from insomnia. Shorter rapid eye movement (REM) latency is particularly prominent, and this also correlates with abnormal DMS and TRH responses.

Electroconvulsive therapy is the oldest and probably the most effective treatment for severe depression, further suggesting that disturbances in the brain's electrical activity may play a crucial role in depression. Spontaneous seizures were originally treated by stress reducing agents such as barbiturates and bromides until the advent of phenytoin (Dilantin). Although this was the first specific anticonvulsant, it was also noted to improve mood and behavior in many depressed patients, and to also provide benefits for patients with arrhythmias and other electrical conduction defects in the heart. Since then, other anticonvulsant drugs such as Depakote, which promote electrical stabilization in the brain, have been found to be even more effective in the treatment of bipolar disease, and may have unusual potential for other types of depression.

These observations furnish more questions than answers with respect to solving the enigma of depression. However, they do augur a new approach to its classification and treatment based on scientific standards that can be measured, rather than current criteria that tend to be both subjective and speculative. (See Book Review on back cover for further discussion.)

Paul J. Rosch, M.D., F.A.C.P.
Editor

More On Stress And Diabetes

It is estimated that about 13 million Americans have type II or non insulin dependent diabetes. Since this is associated with aging, type II diabetes is becoming increasingly common, and now affects 15% of all Americans over the age of 65. It may have several causes. In some patients, the pancreas does not secrete enough insulin in response to an elevated blood sugar. In others, insulin secretion

may actually be greater than normal, but the body develops a resistance to insulin and blood sugar doesn't fall in response to increased insulin levels. Both of these problems can be treated by controlling the diet and reducing weight (which lowers insulin resistance), as well as the use of medications that either stimulate insulin secretion, or reduce insulin resistance. Type I, or insulin dependent diabetes, appears to be an autoimmune disorder in which the insulin producing cells in the pancreas are attacked and destroyed by the body's immune system. Approximately one million Americans suffer from this condition, which is fatal, unless treated with insulin.

Numerous reports in humans and experimental animals confirm that emotional stress causes a rise in blood sugar and also increases the requirements for insulin or other medication in diabetic patients. Stress can also adversely affect diabetic control by encouraging faulty eating habits and disrupting regular diet and meal schedules, or consistency in taking required medication. Some studies have demonstrated a relationship between the magnitude and frequency of life change events and the onset of both type I and type II diabetes. Stress worsens diabetic control in all diabetics because it elevates blood sugar and increases insulin resistance. Conversely, stress management training may result in significant improvement. Successful results have been reported following the regular practice of progressive muscular relaxation, meditation, EMG, biofeedback, yoga, and even cognitive approaches such as rational-emotive therapy. This may be due to a reduction in stress related hormones that elevate blood sugar. Exercise often improves diabetic control because it helps to burn up calories and reduce weight. However, for many joggers, the stress reduction benefits of regular running may result from its stress reducing attributes.

Psychosomatic Medicine, 55:380, 1993

Man may be the captain of his fate, but he is also the victim of his blood sugar.

Wilfred G. Oakley

Stress, Cardiovascular Disease, And Insulin Resistance

Hypertension is a prominent "risk factor" for heart attacks. However, if your blood pressure is elevated, lowering it will not significantly reduce your chances for suffering a coronary event. Although hypertension is associated with heart attacks, it really doesn't cause them. Similarly, it's long been observed that diabetes, hypertension, heart attacks, obesity, diabetes, elevated cholesterol, abnormal blood lipid patterns and accelerated atherosclerosis all seem to go together. If you have one or two of these, it's more than likely that some of the others will eventually appear, so what's the connection? The common link may be due to a phenomenon known as insulin resistance. This refers to a defect in glucose metabolism that leads to the production of increased amounts of insulin. It is only fairly recently that techniques have been developed that have permitted insulin resistance to be measured with accuracy. This has now confirmed a strong correlation between the degree of insulin resistance and levels of blood pressure in patients with coronary heart disease. Stress is also associated with an increased incidence of hypertension, coronary heart disease, diabetes, obesity, elevated lipids, etc., and it appears that some of these relationships may also be mediated through effects on insulin resistance.

To investigate this possibility, Swedish investigators studied the effect of mental stress on insulin sensitivity in male and female diabetic patients who required insulin. The degree of mental stress was not particularly severe. It was provided in the form of a 20 minute modified film version of Stroop's color-word conflict test, in which a color is shown but is described incorrectly by the accompanying text. Insulin sensitivity and resistance was measured, and assessments were also made of cardiovascular and stress-related hormone function. There was a significant increase in insulin resistance during this period of sudden but comparatively mild mental stress as determined by measurements of glucose and insulin in response to various stimuli.

Blood pressure and heart rate also rose during the periods of mental stress, as did levels of cortisol, growth hormone, and adrenaline.

The transient rise in blood sugar, heart rate and blood pressure seen during acute stress have been attributed to increased sympathetic nervous system and adrenal medullary and cortical hormone effects. Although patients with chronic hypertension and/or diabetes do not show evidence of increased adrenal or sympathetic nervous system activity, they do consistently demonstrate increased insulin resistance. It now seems likely that stress induced increased insulin resistance may partially explain how it can contribute to diabetes, hypertension and heart attacks.

Internal Medicine World Report-August 1993

Stress And Bulimia

Bulimia is a disorder characterized by episodes of binge eating followed by self induced vomiting or purging with laxatives. It is associated with neuroendocrine alterations similar to those found in patients with anorexia nervosa and obsessive compulsive behavior, both of which are often stress related. Vasopressin levels are frequently elevated in patients with anorexia and obsessive compulsive disorder, and a recent study now reports increased levels in the spinal fluid of bulimic women.

The significance of this is not clear. Vasopressin is a posterior pituitary hormone, best known for its ability to cause the kidneys to retain water, and is commonly referred to as the antidiuretic hormone. However, animal studies have also revealed that it has the ability to prolong memory for learned associations. Patients with anorexia and bulimia have a very distorted sense of body image. Although often painfully thin, they perceive themselves as being grossly overweight and unattractive. Researchers suspect that starving, avoiding food, or binge-purge cycling, represent tactics that become learned associations with this perception of being overweight. Vasopressin may entrench and prolong memory for this associative link, and thus reinforces these behaviors to prevent weight gain.

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Studies using vasopressin nasal spray in humans also reveals that it improves memory and concentration skills.

Why should vasopressin have anything to do with memory or learning? One explanation is that its markedly increased secretion during acute stress might improve decision making and memory for life saving activities required for future use when confronted with similar threats. Unfortunately, this property could also contribute to the persistence of abnormal behaviors in stress related eating and obsessive compulsive disorders. There may be other links to bulimia. Vasopressin causes the anterior pituitary to release ACTH, which in turn stimulates the adrenal cortex to make cortisol and other hormones. Cortisol levels are also invariably elevated in anorexia nervosa and bulimia, and some newer treatment approaches for these disorders are based on correcting this abnormality. The role of the posterior pituitary in the response to stress has only recently begun to be appreciated. In addition to vasopressin, it releases oxytocin, a hormone used to stimulate the uterus to contract during labor and to increase post partum milk production. Oxytocin levels are generally suppressed during stress, which, as noted previously, is not surprising. It has been found to stimulate sexual desire and activity when injected into non pregnant laboratory animals of both sexes, but making love would probably not be uppermost in one's mind when the only viable choices are "fight or flight".

Science News-April 1992

Let the first satisfaction of appetite be always the measure to you of eating and drinking; and appetite itself the sauce and the pleasure.

Epictetus

Treating Diabetes With Yoga

Since stress elevates the blood sugar and can aggravate diabetes, it would seem plausible that stress reduction strategies might be beneficial for some diabetic patients. A recent study attempted to

examine this using yoga, which has been practiced in various forms in the Orient for centuries. Almost 150 non insulin dependent diabetics were instructed in specific yoga techniques which they practiced for ninety minutes in the morning and an hour in the evening, every day, for six weeks. These included body postures (Asanas), breathing exercises (Pranamayama) and visceral cleaning procedures (Shatkriyas). Fasting blood sugar and glucose tolerance tests were performed before and after the period of yoga therapy.

More than two out of three patients showed fair to good improvement in these tests, and there was a significant reduction in the amount of medication required to control their diabetes. Although there was no change in the daily total number of calories, the yoga diet is primarily vegetarian. Such diets are apt to be rich in complex carbohydrates, which might also have helped to improve diabetic control. However, this study is consistent with other research showing that yoga can lower blood sugar, both in diabetics, as well as healthy individuals, and some authorities believe that this is largely because of its stress reduction properties.

Diabetes Research Clinical Practice, 19:74, 1993



"I don't go to the doctor anymore. He's started to prescribe exercise instead of bed rest."

Doubts About Diet, Cholesterol And Heart Attacks

The lipid hypothesis dominates the field of atherogenesis largely due to "strong epidemiological evidence" of the links between dietary fat, blood cholesterol levels and coronary heart disease (CHD). Some of the numerous fundamental flaws in this approach include the following half dozen considerations:

1. CHD is not a specific disease but a manifestation of many disorders. The correct parameter requiring quantification of atherosclerosis is its severity, which cannot be measured during life. CHD and atherosclerosis are often used as synonyms in clinical trials where CHD is used in some, and angiography or ultrasound in others, to evaluate atherosclerotic severity.

2. Although statistical associations never prove cause and effect, more than 270 associations with CHD are classified as risk factors. However, instead of being used in the correct actuarial sense to indicate the likelihood of development of CHD, the term risk factor is commonly assumed to represent causality, as evidenced by current concepts of "prevention of CHD".

3. In clinical studies, it is often the incidence of risk factors that is compared, rather than correlation of their duration with CHD, even though the correlation should be with severity of atherosclerosis.

4. Subjects in clinical trials are usually under the age of 55, while over 80% of those dying of CHD are over 65, leading to selection bias for familial hypercholesterolemia (FH). The majority of male heterozygotes, but fewer females, suffer heart attacks before the age of 50. This gives a false impression of hypercholesterolemia for CHD, since mean values are generally used for a group. The number of FH participants is rarely given, but they should probably be excluded, since the pathogenesis of their heart attacks differs from conventional CHD of atherosclerotic origin.

5. Epidemiologists rely heavily on monocausal mortality rates although studies show that the diag-

nosis of CHD is often based on probability, and that false positive rates range from 14% to 76% and false negatives from 20 to 85%.

6. The accuracy of assessment of dietary intake is notoriously low, and extrapolations from a week's or month's diet by recall cannot be considered to accurately reflect the intake of such constituents over 40 or 50 years.

Stress has a much more powerful effect on serum cholesterol levels than dietary fat intake. Its contributory role to coronary heart disease is more likely mediated by neuroendocrine pathways that produce direct damage to the cardiovascular system and other alterations, than elevations of serum lipids.

Adapted from *Diet, Cholesterol, And Heart Disease: Epidemiologic Illusion Or Delusion?*

Paul J. Rosch - The American Institute of Stress, Yonkers
Fifth International Montreux Congress on Stress

More Frogs Are Croaking Due To Stress

"Something is killing the world's frog population, and it just might be stress". That's the opinion of some scientists who attended the recent annual meeting of the Society for the Study of Amphibians and Reptiles. There is little doubt that there has been a progressive and significant decline in numerous species of frogs throughout the world. In selected areas, natural fluctuations and man made problems such as acid rain or destruction of natural habitats might be a factor, but increased stress appears to be the best explanation. Researchers believe that "something is stressing the amphibians' immune systems, enabling diseases to turn lethal.... If stress is the answer, finding the cause may be very important to the world's human population." As one biologist stated, "We see frogs as the first indication that something is wrong out there. If we don't pay attention to it, we could be very sorry." One surprising bright spot has been the Midwest flooding. "Much as you hate to say it, it's been a wonderful year in the Midwest for frogs."

Associated Press 9/9/93

Predicting Mortality In Coronary Heart Disease

Considerable research efforts have been devoted to demonstrating the role of behavior and personality factors as contributors to coronary heart disease. The Type A Behavior hypothesis is one example of this type of approach. Along with others, it may have consumed a disproportionate part of research resources, since results and conclusions have been largely contradictory and inconclusive. Instead of focusing solely on possible behavioral factors, we suggest that investigation of interactive psychosocial influences may prove more productive. This includes interrelationships between the impact of the social environment, individual psychological and behavioral reactions, life styles and psychophysiological responses.

We have utilized such an approach in a study of prognostic factors in 150 male cardiac patients, who were followed for ten years. Type A Behavior ratings alone did not affect their survival, but when combined with a lack of social integration and cohesion and psychological responses characterized by depressive reactions, it increased the mortality prediction considerably. Men who were both Type A, depressed and socially isolated had the highest mortality rates. This risk was independent of other relevant factors such as the degree of myocardial damage, cardiac enlargement or increased cardiovascular as assessed by dysrhythmia on ambulatory ECG-monitoring. This biopsychosocial integrative approach significantly increased our ability to predict mortality risk in male patients with coronary heart disease. Further research is required to determine its applicability and efficacy in other stress related disorders.

Abstracted from *Social Ties And Cardiovascular Health*
Kristina Orth-Gomer - Karolinska Institute, Stockholm
Fifth International Montreux Congress On Stress

It is a test of true theories not only to account for but to predict phenomena.

William Whewell

Sudden Death And Life Change Events

Cross-cultural studies of cardiac patients who succumbed to sudden death, revealed a three-fold increase in recent life change experiences in the victims' final six months of life, compared to estimated baseline levels. These life change events occurred in areas of work, home and family life, personal and social life, finances, and health. No single area was found to predominate.

Cardiac patients who survived a myocardial infarction showed a two-fold increase in build-up of recent life changes over the six months prior to their infarctions. (To control for possible reporter bias, both sets of data were collected from the next-of-kin of all cardiac patients.) It was concluded that the psychophysiological arousal secondary to patient's recent life change experience bears a direct relationship to outcome following a myocardial infarction. In other words, the greater the magnitude of recent life change increases experienced by coronary heart disease patients, the greater the risk for sudden cardiac death.

A retrospective study of all deaths in Finland over a one year period similarly revealed a background of "stressful" events during the 6-month period prior to the terminal event of each of the sudden cardiac death victims. This form of "stress" was not evident during the antecedent interval in controls dying from other causes. Bereavement, the most highly rated life-event linked to sudden cardiac death, is a "stressor" that appears to be uniquely human. Other relevant life-events were identified which also seem to require the participation of higher brain functions, such as job insecurity and marital strife. However, life-events that could be shared by the higher and lower animals (e.g., recently moving to a new environment, a "novelty stress") were also important. Thus, both higher and lower cognitive mechanisms appear to be involved in the biological mechanisms(s) underlying sudden cardiac death.

Abstracted from *Recent Life Change Regulates Sudden Cardiac Death*

Richard H. Rahe - Nevada Mental Health Institute, Sparks
Fifth International Montreux Congress on Stress

Book Reviews • Meetings and Items of Interest

Book Review

The Fundamentals of Psychopharmacology, B.E. Leonard, John Wiley & Sons, Ltd. Chichester, 267 pages, \$32.95.

This is an excellent and much needed contribution. Its value can best be appreciated by a realization that a meaningful book with this title could not have been written a few decades ago. Since then, considerable progress has been made in the treatment of specific disorders due to new classes of pharmaceuticals. But how are drugs for mental disorders developed? When it comes to medical problems like diabetes or hypertension, experimental animal models are readily available. However, there are no animal models for schizophrenia, depression, mania, or obsessive-compulsive disorder. Selecting or designing effective drugs can only be based on an understanding of how they may correct biochemical abnormalities that may underlay certain symptoms or behaviors.

Professor Leonard's book goes a long way in filling a large void in this area. It contains a very thorough discussion of the basic aspects of neurotransmitter function and how these chemical messengers exert their psychophysiologic effects. Chapters are devoted to the psychopharmacology of depression, mania, anxiety disorders, insomnia, schizophrenia, epilepsy, Parkinsonism, and Alzheimer's disease. Specific chapters also deal with the special problems associated with the use of psychotropic medications in pediatric and geriatric patients. Another deals with the complex subject of pharmacokinetics, and the significance of monitoring drug therapy based on blood levels. A lengthy chapter is devoted to the psychopharmacology of all drugs of abuse ranging from alcohol and PCP, that cause problems of dependency and addiction.

Charts and figures are attractively presented and are extremely helpful in explaining complex topics

clearly. Appendices contain Tables describing important psychotropic drug interactions, a Glossary of common terms used in psychopharmacology, listings of generic and brand names for all psychotropic drugs, and references and suggested readings for each chapter. This book is chock full of useful information. It should be required reading for all psychiatrists and those who prescribe psychotropic agents, and anyone remotely interested in the biochemical basis of mental illness.

Meetings and Items of Interest

October 21-24 Energy Medicine and Body/Mind/Spirit Integration, A National Association of Holistic Healing Conference, La Jolla Marriott, San Diego, CA (804) 422-9033

October 28-31 The 6th National Conference of the Society for Professional Well-Being, Being Well: Adapting to Professional and Personal Change, Menger Hotel, San Antonio, TX, contact Marjorie Harrison (919) 489-9176

October 29-31 Harvard Medical School, Massachusetts General Hospital, Department of Psychiatry - Treating Personality Disorders, Westin Hotel, Boston (617) 432-1525

November 11-14 Academy of Psychosomatic Medicine 40th Annual Meeting: Bridging the Gap Between Knowledge and Clinical Practice - The C-L Psychiatrist's Role, The Fairmont Hotel, New Orleans, LA, for info (800) 447 4474

December 1-12 5th International Conference, The Psychology of Health, Immunity and Disease, Hilton Head Island, SC (800) 743-2226

January 9-11 Mental Health, Substance Abuse, Managed Health Care, The Fontainebleau, Miami, FL (202) 778-3200

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