

The Newsletter of THE AMERICAN INSTITUTE OF STRESS

Number 2

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STRESS AND STRAIN: Does It Really Cause Coronary Heart Disease?

That was the title of the paper I was asked to present at the recent Sixth International Montreux Congress On Stress, organized by The American Institute Of Stress and Biotonus Clinic. I had asked my good friend, Ray Rosenman, to organize a full morning session devoted to "Psychosocial Concepts in Cardiovascular Physiology", as he had done in previous years. He agreed, with the proviso that I would provide a convincing case for the role of stress in coronary heart disease in a 20 minute presentation based on this title. He laid down certain ground rules, emphasizing that my discussion could not refer to either sudden death or congestive failure. I also suspected from past conversations with Ray that Type A behavior would similarly be off limits. It would be comparatively easy to make a case for incriminating stress in the precipitation of sudden death or congestive failure, and there is abundant support for the role of Type A Coronary Prone Behavior. However, strictly speaking, Type A behavior really has very little to do with stress, despite

considerable confusion about this on the part of the public and the profession.

On the other hand, these various proscriptions serve to illustrate and emphasize the semantic confusion that surrounds the subject, verb, and object of the title that was assigned to me. The residents of Babel used different words for the same thing, because they spoke in different languages. However, we often use the same word to refer to very dissimilar things, which is just as bad. There is a natural but unfortunate tendency to assume that just because you have given something a name, that you now somehow have defined it, or worse, possibly understand what it means or even signifies. This applies not only to "stress" and "strain", but also "cause", and certainly "coronary heart disease".

Stress: A Semantic Snake Pit

I am sure we can all agree that there is no satisfactory scientific or objective definition of "stress". Selye, who coined the term as it is currently used, originally defined it as "the non specific response of the organism to any demand for change", but this proved somewhat vague, and had little significance for most people. "Stress" had been used for several hundred years in

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

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physics to denote an external force which acts on a resistant object in an attempt to deform it. "Strain" is the resultant distortion or changes that are produced, as in Hooke's Law. The ratio of stress to strain is a characteristic property of a material called the modulus of elasticity. This value is high for a rigid material like steel, and much lower for malleable metals.

Actually, Selye was really referring to strain, and he once confided in me, that had his knowledge of English been more precise, he would have gone down in history as the father of the "Strain" concept. He had a lot of trouble keeping to his own definition, which created further confusion. In 1951, when we were preparing *The First Annual Report on Stress*, I cited one critic, who complained in the *British Medical Journal* that "according to Professor Selye, stress, in addition to being itself, is also the cause of itself, and the result of itself". This was based on verbatim citations from Selye's massive tome *Stress*, which had been published the previous year.

This problem still persists. Stress is often used to refer to some unpleasant threat (being held up at gun point), a psychophysiological response (panic or palpitations), or the presumed pathological consequence of this interaction (peptic ulcer or heart attack). This semantic confusion created all sorts of problems when Selye's research had to be translated

into French. That is how *le stress* was born, and was quickly followed by *el stress*, *il stress*, *der stress*, and *lo stress* in other European languages, with similar neologisms in Russian, Japanese, Chinese, and Arabic. Stress is one of the very few English words you will see preserved extant in scientific publications in all languages. Selye subsequently had to create another new word, "stressor", to clarify the distinction between stimulus and response. Selye recognized the muddle and disarray that existed in the field, much of which he had created and constantly struggled to find some solution. In a book written for the general public two decades after his initial description, he acknowledged how confusing the term *stress* had become. In an attempt at clarification, he suggested this revised definition.

"Stress is the state manifested by a specific syndrome which consists of all the specifically induced changes within a biologic system. Thus, stress has its own characteristic form and composition but no particular cause."

He listed ten things that stress was not, including nervous tension, the secretion of stress-related hormones, anything that caused an Alarm Reaction, a disturbance of homeostasis, etc. However, this was quite contrary to what most people believed, and simply created more confusion.

We customarily use the word stress as a variant or shortened form for distress. However, it can also refer to pleasurable stimuli or events, which Selye called "eustress". Winning a race or an election can be just as stressful as losing, or even more so. Sudden death due to stress can follow news of the unexpected death of a loved one, but also occurs in young brides overcome by excitement on their wedding day. Thus, stress is a useless word for some scientists because it cannot be adequately defined, much less measured. Nevertheless, it somehow best embraces the variegated phenomena that fall under the broad heading of mind-body relationships, and stress will not likely be replaced by any other word or phrase that is more meaningful or accurate.

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What Does Cause Mean?

The verb in the title of my assignment, "cause", presented similar problems. Exactly what do we mean when we say that something **causes** something else. At the crux of this issue, is the meaning and use of cause as it relates to signs and symptoms of illness, as opposed to a specific pathologic process. In order to comprehend this, it's necessary to have a very clear concept of what a specific disease entity constitutes and not to use the word disease to refer to various signs and symptoms, such as chest pain, elevated blood pressure, or high cholesterol. Even clinical diagnoses such as hypertension, stroke, and heart attack, do not represent specific diseases. Essential hypertension is not a discrete diagnosis. It is simply the description of a persistently elevated blood pressure that could have many different origins. Unless you can be certain of the source of hypertension in any given patient, you do not know its cause, and this also applies to coronary heart disease. While it is obvious that every disease must have a cause, it is essential to distinguish this sharply from a **contributing** factor. Prevention and proper treatment depend upon identifying the *causa vera*, or "true cause", which is specific.

The germ theory, Koch's postulates, and the discovery of vitamin deficiency disorders, contributed tremendously to our understanding of the *causa vera* of certain disorders. It's impossible to develop tuberculosis without being infected by the tubercle bacillus, or to develop scurvy unless there is a lack of vitamin C. However, in the last century, it was generally believed that tuberculosis was due to close, unsanitary living conditions. Support for this came from the observation that when such situations were corrected, there was a dramatic reduction in tuberculosis. This belief persisted until Koch identified the tubercle bacillus as the *causa vera*, which was probably the first demonstration of a specific relationship between a microorganism and an infectious disorder. For prevention and treatment to be completely effective, it is necessary to eradicate the *causa vera*, or "true cause" of tuberculosis, rather than associated findings, signs and symptoms, such as unsanitary living conditions, fever, and hemoptysis. With respect to coronary heart disease, this also applies to hypertension, elevated blood cholesterol, cigarette smoking, stress, and chest pain.

It is also very important to emphasize that the presence of the *causa vera* does not always guarantee that disease will result. A good example of this is afforded by diseases known as "the fevers", that swept across Europe in the latter half of the last century. Although Koch subsequently identified vibrio cholera as the major culprit, others, like the hygienist Max von Pettenkoffer, doubted that the bacillus by itself could cause cholera. To prove this, he grew a fresh culture of the organism from material collected from a fatal case in the current Hamburg epidemic, and swallowed a large amount, which would have been considerably in excess of any normal exposure. He did this on an empty stomach whose acidity had previously been neutralized by ingesting a sufficient amount of sodium bicarbonate, since it had been shown that these were the most favorable conditions to promote the growth of this organism. Although large numbers of active bacilli could be cultured from the stool, he experienced no adverse symptoms other than mild diarrhea.

This is an important consideration for those interested in stress related disorders, since in many instances, the mechanism of action may be mediated by lowering host resistance. Sir William Osler believed that stress played such an important role in tuberculosis, that it was more important to know what went on in a man's head, than in his chest, to predict its clinical course. Active infectious bacteria and viruses can be cultured from many individuals who show no evidence of illness. In one well documented recent study, several hundred volunteers received nasal drops containing large doses of one of five respiratory viruses. Stress levels were evaluated by scores obtained from combining three standard measures that included severity of current negative emotions as well as life change events over the preceding year. Infection rates as assessed by specific antiviral antibodies ranged from 75-90 percent, but clinical colds occurred in much less than half of these. The startling finding was that rates for both laboratory evidence of infection and clinical disease correlated precisely with the magnitude of psychological stress scores for each of the viruses. Would it be correct to conclude, therefore, that these colds

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were caused by stress?

We need to agree on what "cause" really signifies. If we say A causes B, does it mean that you can't have B unless you have A? Does it mean that every time you have A, B will result? If we were to ask the general public, or even physicians, "**What causes heart attacks?**", the answers would most likely be severe coronary atherosclerosis, high cholesterol levels, hypertension, cigarette smoking, strong family history, diabetes, and some might suggest stress or Type A behavior. However, heart attacks, can occur in the absence of all of these. Therefore, it is critical and crucial to make a sharp distinction between true causes, as opposed to associated findings, especially when discussing coronary heart disease.

What Is Coronary Heart Disease? Could It Ever Be Contagious?

Exactly what do we mean when we refer to coronary heart disease? The term is often used interchangeably with, or to signify, angina, coronary insufficiency, myocardial infarction, coronary occlusive disease, sudden death, specific electrocardiographic alterations, findings on various imaging procedures, etc., etc. However, it's possible to have electrocardiographic changes consistent with significant coronary ischemia without any signs or symptoms that would suggest such a problem. It's also possible to have extensive occlusive coronary atherosclerosis in the absence of any relevant signs, symptoms, or even electrocardiographic changes. Anginal pain may be due to coronary vasospasm without significant obstructive disease, coronary occlusion can occur without myocardial infarction, and myocardial infarction can occur without a coronary occlusion. Stress related sudden death due to ventricular fibrillation is usually not associated with evidence of either acute coronary occlusion or myocardial infarction. However, all of the above are often referred to as evidence of "coronary heart disease", or are assumed to be due to advanced coronary atherosclerosis.

Most often, coronary heart disease is used synonymously with, or to signify, coronary atherosclerosis, but this is also incorrect. Is coronary atherosclerosis the same as atherosclerosis elsewhere in the body? Is atherosclerosis a distinct and specific disease? If so, then its true cause must be identified as having been present in every individual or animal with atherosclerosis, as we could demonstrate for the tubercle bacillus and tuberculosis. In addition, the occlusive atherosclerotic lesions seen in familial hyperlipoproteinemia, or in animals on high cholesterol diets, are quite different from the atherosclerotic plaque commonly associated with coronary morbidity. They do not have the characteristic foam cells and other distinctive inflammatory stigmata. It has been suggested that in certain patients, various microorganisms could cause coronary heart disease by promoting the development of inflammatory atherosclerotic lesions. Indeed, some authorities view atherosclerosis as a chronic, low grade arterial infection which is aggravated by hypercholesterolemia and other risk factors. They suggest that there are probably multiple potential infective pathogens and routes of transmission that might initiate atherosclerosis, including numerous viruses that produce clinically silent infections in animals. Pathways for transmission to humans might be via food, which could account for the parallel increases of meat consumption and mortality from coronary heart disease seen in the U.S. during the middle third of this century. Atherosclerotic plaque may result from infection with cytomegalovirus and other herpes viruses, based on antibody studies and transplant atherosclerosis findings. A Finnish study suggests that a strain of chlamydia encountered in common upper respiratory infections may also be involved. Researchers found that 60% of heart attack patients had antibodies to this strain of chlamydia, which is spread by droplet infection, in contrast to only 20% of a control group.

Coronary heart disease is a very imprecise and inappropriate monitor of the severity of coronary atherosclerosis. The problem is further compounded by the inapplicable extrapolation of coronary heart disease risk factors as being causes of coronary heart disease and/or atherosclerosis, rather than merely statistical associations.

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Association Is Not Causation

The Framingham research demonstrated in retrospective and prospective studies that heart attack patients tended to have higher cholesterol, blood pressures, and smoked more. The obvious conclusion appeared to be that if you could eradicate or reduce these "risk factors", that this would significantly lower the incidence of subsequent coronary events. This was not achieved in the massive, multicenter MRFIT study, where coronary mortality was higher in treated hypertensives than untreated controls. Two other large trials in Finland and Europe, similarly showed no benefits from reducing these risk factors. **Risk factors are not causes of disease, but merely statistical associations, and you can't use a statistic to prove another statistic.** A more accurate and appropriate designation for smoking, hypertension, and cholesterol, would be "risk marker".

This also applies to stress. I could have satisfied my assignment by citing the role of stress in promoting all the common risk factors for CHD. Stress contributes significantly to hypertension, elevated serum lipids, and cigarette smoking. I might have pointed out the increased incidence of coronary morbidity and mortality associated with such psychosocial stresses as poverty, loneliness, rapid sociocultural change, crowding, and having little control over one's life. There would have also been support from the salubrious benefits that stem from the stress buffering effects of a strong social support system, firm faith, and other examples of eustress. However, the bottom line is that we don't know what stress is, we don't know what strain is, it's not clear what cause means, or what coronary heart disease really refers to. Perhaps some day, we will have the tools and skills to demonstrate that stress and strain can cause coronary heart disease. But this will never happen until we all agree on exactly what these terms mean.

Does Stress and Strain Really Cause Coronary Heart Disease? At present, I suspect the best answer is "perhaps", since the evidence for "stress" is as strong, -and as weak-, as anything else that has been proposed. The message I wished to convey at the Congress was that **association should never be confused with causation.** Stay tuned.

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

Reducing Stress Improves Congestive Failure

Congestive heart failure is a condition in which the pumping efficiency of the heart is diminished and the lungs and peripheral tissues become overloaded with fluid, leading to shortness of breath and ankle swelling. Drugs like digitalis to improve the efficiency of heart muscle contraction, diuretics to remove excess fluid, or vasodilator to reduce blood vessel constriction may help, but often have undesirable side effects that interfere with quality of life. Prior studies have shown that stress reduction and biofeedback can reduce the arterial spasm seen in patients with Raynaud's disease and hypertension, often eliminating the need for drugs.

A new study suggests that such stress reduction approaches can also significantly improve circulation and reduce shortness of breath in patients with congestive heart failure. Patients were told to concentrate on relaxing scenes for 20 to 30 minutes, and cardiac output, respiratory rate, skin temperature, oxygen consumption, and stress hormone levels were measured before, during, and after this session.

In the 25 patients studied, at least 1 out of 5 experienced a significant decrease in vascular resistance, and more than 1 out of 4 had an improved cardiac output. Skin temperatures increased an average of 3 degrees, indicating greater relaxation in peripheral blood vessels. Respirations decreased more than 3.5 breaths per minute.

The fact that these changes could be demonstrated with just one relatively short session is quite impressive. While not everyone will respond to this type of intervention, it may be possible to select individuals who would be most likely to benefit from stress reduction. In addition, multiple sessions may be even more beneficial, and a new study is underway which will include 6 weeks of biofeedback and relaxation to demonstrate this.

Medical Tribune-September 8, 1993

People who have no weaknesses are terrible; there is no way of taking advantage of them.

Anatole France

Mental vs Physical Stress and Your Heart

Which is worse? Obviously, that would depend on how much and what kind of stress you are subjected to, prior physical conditioning, and a variety of other considerations. The effects of physical stress are well known, and standardized treadmill exercise tests are routinely used to measure cardiac function. However, the significance and consequences of mental stressors are much more difficult to evaluate, since their magnitude cannot be quantified with any meaningful degree of accuracy.

In an attempt to examine the relative importance of emotional stress in patients with coronary heart disease, 50 patients were selected in whom electrocardiographic evidence of ischemia was provoked by mental or emotional stress. This was induced either by performing mental arithmetic calculations, reading out loud, or delivering an emotionally arousing speech dealing with some personal issue. Responses were then compared to those which were seen in standard treadmill exercise stress tests.

Sixty seven per cent developed electrocardiographic evidence of ischemia during mental stress tasks, compared to 29 to 76.3% undergoing treadmill stress testing. The personally relevant, emotionally arousing speech challenge caused significantly more frequent changes than the others. In addition, the magnitude of these ischemic changes was similar to that seen during treadmill testing. Reading tasks provoked more ischemia than mental mathematic trials.

In general, electrocardiographic changes tend to occur during treadmill testing as heart rate and/or blood pressure rises significantly. Of particular interest in this study, was the finding that in those patients in whom both exercise and speech tasks produced ischemic changes on the electrocardiogram, blood pressure and heart rate increases were quite different at the time these abnormalities occurred. Ischemic changes occurred at significantly lower heart rates and blood pressures during the

emotionally arousing speech task than during treadmill stress testing.

This study may have important implications. It confirms that emotional stress can be just as important as physical stress, with respect to the production of ischemia, and suggests that the mechanism of action may be different. It also implies that emotional stress may be more important in the production of "silent ischemia", since such patients are much less likely to recognize they are at risk, in the absence of hemodynamic clues.

J Assoc Physicians India-February 1993

The heart has its reasons which reason does not know.

Pascal

The Health Benefits of Music vs Meditation

The stress reduction effects of music have been known since antiquity. Saul was soothed by David playing the lyre, and a few centuries ago Congreve wrote that "music hath charms to soothe a savage breast". Regular meditators often report that they can think more clearly, and occasionally can enter a state of deep relaxation but heightened mental acuity that facilitates problem solving. It had previously been reported that music participation courses could improve the thinking ability of preschoolers, and the same now appears to be true for college students, at least when the music is Mozart. Thirty six subjects were given intelligence tests after listening to ten minutes of Mozart's two piano, D major sonata, and equivalent periods of time listening to relaxation tapes or practicing meditation. IQ scores averaged 9 points higher following listening to Mozart compared to either of the other stress reduction activities. However, this effect was apparently not sustained, since those who listened to the music as the first part of the three phase experiment did no better after the combination of the three were completed, than those for whom the musical selection was last. It is also not

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clear whether or not longer periods of time for each of the three stress reduction modalities selected might have provided different results.

The experiment was conducted in college students by The Center for the Neurobiology of Learning and Memory at the University of California, and it is not known whether older age groups would show similar results. It has been suggested that listening to this type of music may "facilitate certain complex neuronal patterns and stimulate higher brain activities much like math and chess". However, all types of music may not produce these effects. According to the senior investigator, simple and repetitive music might have the opposite effect. Record stores in one major U.S. city allegedly sold out of the Mozart selection the day after the findings were reported in a letter to *Nature*.

Nature-October 1993

Brain Mind Bulletin-October/November 1993



"He must be improving. I'm down to one tranquilizer a day."

New Breast Cancer Study Supports Job Stress Link

As noted in prior issues of the Newsletter, there has been an alarming increase in breast cancer in middle-aged women, for which there is no apparent explanation. We suggested several years ago that psychosocial stressors and particularly job stress might be a major factor. Career oriented women tend to marry and become pregnant later in life, or never become pregnant at all. Pregnancy reduces levels of prolactin, a pituitary hormone known to

promote breast tissue growth and mammary cancer in experimental animals. Upwardly mobile female workers are also less likely to enjoy the stress buffering social support benefits afforded by close marriage, family, and personal relationships.

It has been well established that stress can lower immune system defenses to cancer. Women also tend to be subjected to greater degrees of stress in the workplace. Despite equal or superior talent and experience, they are usually paid and/or promoted less than male counterparts. Female executives encounter a glass ceiling when they try to climb the higher rungs of the management ladder, and sexual harassment and discrimination occurs at every level.

A new survey of almost 3 million women who died between 1979-1987 now confirms much higher than expected breast cancer mortality rates among executive professionals and administrative workers. The study, conducted under the auspices of The National Institute For Occupational Safety and Health, indicates that these findings cannot be explained by any common workplace exposure. Professional women who delay childbirth during education and career building, or who remain childless, may suffer a 50-100% greater than expected risk of dying from breast cancer, according to the CDC report. Among white women, breast cancer mortality rates for women clergy, librarians, and teachers, were 50% higher than white women in general, and black teachers had twice the risk of breast cancer death than other blacks. In 1988, its statistics revealed that in women with 5 or more years of college, 36% of births occurring at age 30-44 were first births, compared to only 16% of women with a high school education. Nearly a third of female managers and professionals giving birth at ages 30-44 were having their first child. In contrast, only about a fifth of women in service occupations who gave birth at that age, were having their first child.

The Wall Street Journal-September 15, 1993

STRESS: the feeling you get when your gut says "No" and your mouth says, "Yes, I'd be glad to."

Dick Francis

Book Reviews • Meetings and Items of Interest

Book Review

Principles And Practice Of Stress Management Second Edition, eds. Paul M. Lehrer and Robert L. Woolfolk, Guilford Press, New York, 1993, 621pgs., \$65.00

In the nine years since the first edition of this book, there have been numerous new developments in stress research. The editors have done a masterful job in reviewing these and also in updating topics previously covered. Unlike the previous volume which consisted of thirteen chapters devoted to various topics, this book contains some nineteen relevant contributions from distinguished authorities, and is divided into three sections. The first is an Introduction, and discusses distinctions between research and clinical applications, and various philosophical and socio-cultural aspects of stress management. Section 2 deals with different stress management methods, including progressive relaxation, as originally described, as well as abbreviated methods, yoga, meditation, hypnosis and self-hypnosis, autogenic techniques, the use of biofeedback, the role of respiration, cognitive approaches, stress inoculation training, music therapy, aerobic exercise, and the role of drug therapy. The final section, Integration, written by the editors, provides a skillful overview of stress management that carefully explains which procedures work best in different situations.

The authors of each of the chapters have all been carefully selected, and in most instances are the preeminent authorities in their specific areas of expertise. While there is some overlap, this is obviously unavoidable in an offering of such encyclopedic scope. Although a chapter was devoted to music therapy, there was no discussion of aromatherapy, cranioelectrical stimulation, and other subtle energy stress reduction approaches, such as low energy emission therapy with

the Symtonic device. However, this book should be of inestimable value for anyone interested in any aspect of stress management. As the Foreword notes, "Biofeedback Is Not Relaxation Is Not Hypnosis", and that message is clearly delivered.

Meetings and Items of Interest

April 7-10 Acupuncture Applications in Neurologic Disorders and Pain Management, Sponsored by the University of Arizona College of Medicine, Scottsdale Hilton Resort and Spa, Scottsdale, AZ, for info call (213) 937-5514

April 13-16 Fifteenth Anniversary Meeting, "Cross-Cutting Dimensions of Behavioral Medicine: Visions for the Future", Park Plaza Hotel, Boston, MA, Contact Laura Hayman (301) 251-2790

April 15-17 9th Annual International Conference on The Positive Power of Humor & Creativity, Surviving and Thriving in the 90's, Saratoga Springs, NY, for info (518) 587-8770

April 24 Women's Healthcare: Clinical Perspectives in Natural Medicine, The Stamford Marriott Hotel, Stamford, CT, (206) 623-2520

April 26 Recent Advances in Acupuncture Research, The Center for Frontier Sciences Spring, 1994, Colloquia, Temple University, Philadelphia, PA, call (215) 204-8487

April 28-May 1 Children: Our Ultimate Investment, The Celebration of the Birth Centenary of Aldous Huxley, Wilshire Ebell Theatre, Los Angeles, CA, (213) 461-8976

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