Emotional Factors in Coronary Heart Disease

By Howard B. Sprague, M.D.

ANYONE intrepid enough to discuss the role of the emotions in coronary disease exposes himself to stresses that are asserted by some to be lethal. Thus, when I essayed, Canute-like, a few years ago, to challenge the concept that Western civilization is inevitably emotionally bad for the coronaries, I was told, in print,1 that it was “almost incredible” that I should have considered the terrors of grief, war, suicide, manic depressive insanity, and horrendous primitive taboos (stresses under which man has always suffered) as approaching in deadliness the hazards of our socioeconomic existence; and this in spite of the assertion by other psychosomaticists that fear, anger, resentment, frustration, and depression produce a greater cardiac output for a longer time than standard exercise. Naturally this social disapproval, this failure on my part to be “agin Sin,” has been very hard on me.

My friend, Dr. Louis Katz, has rightly said, concerning this matter of the emotions in atherosclerosis, “... too much has been said on this subject on too little evidence. We should do more research and less talking!” However, he stated that the Committee on Emotions and Cardiovascular Disease of the Chicago Heart Association has proposed that it devote 2 years to exploring the field through a series of conferences, to decide whether or not even to undertake a major study of it.2 I predict that the Committee will decide in the negative, but, at any rate, they must still be in the talking stage and will perhaps allow me a word at this meeting. Dr. Katz has admirably outlined the many questions needing answers.

What, I think, is the difference between the two major schools in this clouded area is really the emphasis upon the relative influence of heredity and of environment, which we all recognize cannot be treated separately. As Hogben says, “no statement about a genetic difference is clear, unless it includes or implies a specification of the environment in which it manifests itself in a particular manner.” And in referring to the “Nurture” versus the “Nature” point of view, he says that the former justifies its position by “arguments which suggest that caterpillars of the cabbage butterfly will take to a mixture of pollen and honey. Their opponents appear to hold that Newton would have written his Principia if he had been born in Tasmania.”

The whole concept of causation is, after all, a weighty business and in the obviously polygenic etiology of coronary atherosclerosis and occlusion one must almost admit the legal definition of cause as phrased in the Workmen’s Compensation Act of New Zealand: “Affirmative finding is not justified because it is possible, or even probable, that the disease was so caused, accelerated or aggravated; but it must be the result of a legitimate inference from the proved and admitted facts.”

My inclination is toward the role of inborn errors of metabolism as preponderant over the influences of environment. Were we to capture that will-o-the-wisp, a satisfactory test to define individual liability to coronary disease, I venture to say that the nonsusceptibles could eat what they wished, work as they wished, and indulge in any of the more appealing vices without cardiovascular retribution.

It seems obvious that much energy is being needlessly channeled into the demonstration that emotional stimuli have an effect upon serum lipids. As Page has shown, studies have been presented indicating that the emotions raise, lower, or leave unaffected the serum cholesterol. There are large individual variations and more work is needed in determining normal levels over long periods of time. This

response, like that of other serum lipids, blood sugar, coagulability and fibrinolysis, or blood pressure, is part of the normal balancing of homeostasis; and it is not at all clear that any of these measure an individual liability to coronary atheroma or thrombosis. Pituitary-adrenal or other hormonal effects upon these, or upon local arterial reactions, are similarly obscure.

Dr. Forrest E. Kendall, in inviting me to present the Duff Lecture, emphasized the clinical aspect of my discussion and I am not attempting to review the literature of the biochemical and central nervous system effects of emotional factors upon the circulation. It is perhaps sufficient to point out that there exists an idiosyncratic, stereotypic reaction to mental stress in each one of us, which I doubt very much is predictable by any presently available test or "profile." Secondly, if some normal constituent of the blood is altered by stress, the chances are that the alteration is "good" for the organism, rather than "bad." Originating, as it does in the phylogenetically oldest part of the cerebral cortex, it must have had some survival value. I say this even facing the criticism of my naive teleology.

Thirdly, it has recently been shown that when persons with high scores in tests for maladjustment or anxiety were retested, their maladjustment or anxiety scores were lower the second time. As the editorial comment on this states "... merely repeat the test and do nothing else, and the disorder, at least as measured by the test, will improve!"4

Attempts to define the coronary-prone patients on the basis of the emotional reactions of known coronary patients compared with so-called "controls" of so-called "normal" individuals seem to me unrealistic. Those reported appear not to have considered somatotype as related to its common temperamental component, nor racial differences relative to overt hyperreactivity to irritation. Certainly my patients with coronary artery disease do not conform so beautifully to what has been called the Hollywood concept of the American business man. Of course if some objective screening device on the emotional level were available, the method could test all individuals of an occupational class and discover the hyperreactors, and such could be followed to see if they developed overt coronary disease more often than others. The trouble is that "stress cannot be defined by the dimension of the load."

An aspect of this problem seems to have been disregarded and to which I would like to summon you. It is commonplace for us, as clinicians, to have patients, or their wives, describe a stressful situation in which the first symptoms of coronary occlusion developed. Similarly, as students, we were taught by Richard Cabot that a patient with an acute illness could always remember something that he had eaten that must have precipitated his attack. I will grant that there are relationships, at least temporal, between some attacks of myocardial infarction, or the first appearance of angina, and a specific event, an event that may have revealed the underlying coronary disease earlier than it might otherwise have shown itself. Aarons recently found only two such claims attributed to exciting arguments in a series of 100 insurance cases with fatal heart attacks. But what I am referring to is not the alleged precipitating event, but rather the theory of prolonged or severe emotional tension, existing for weeks or months before the coronary occlusion as of great etiologic significance.

There does not seem to be much question but that at least 50 per cent of the men in this country have significant coronary pathology by the age of 50 and that we are all exposed to mental stress. For the sake of argument, let us attribute the downfall of the coronary patient to his job stress or what has been called the "general forms of socioeconomic stress unique to Western societies." In this connection let me quote from Parkinson's book "The Law and the Profits" the items of a bill rendered to an Englishman by a tax consultant as follows: "To enable you to evade the income tax payment, a laborious and intricate work, your accounts extending over a period of fifteen months, £ 6.6.0."

The antiquity of these supposedly lethal socio-
economic stresses is demonstrated by the fact that this bill was rendered in 1852, 108 years ago, and long before anyone ever heard of a coronary thrombosis. One wonders if the tax consultants of a hundred years ago had seasonal rises of serum cholesterol and shortened coagulation times or, as Gofman has shown, if they, like so many others, eat more under stress to account for a bit of a rise in blood lipids.

But the point I wish to make is that even if the dominating role of the emotions in atherogenesis or short clotting time is assumed, why is it that continuation of such stresses or even increase in them fails to result in another coronary thrombosis in an impressive number of patients?

The physician by his special brand of necromancy may be credited with protecting his patient from further destruction, be it through advising business adjustments, exercise, low-fat diets, polyethylene fats, or anticoagulants, but if he is reasonably honest, he will recall men who disregarded all his advice and lived merrily on, in spite of the direst of predictions.

In my own experience, it seems to me that the man who recovers from a myocardial infarct sufficiently well to return to his usual work does best if he stops smoking, reduces weight, and increases his physical activity. Indeed, ability to follow such a regimen, I use as a test of his real motivation to get well. But since I am encouraged to be clinical, let me give you a few examples:

1. A building and road contractor of 44, first seen in 1949, who "said he had been working out of business grief" for 4 to 5 years, had vague chest pain for 6 years thereafter until he had a myocardial infarct in 1955. This attack, however, although it was preceded by hard work getting contracts, came after a New Year's Eve party lasting to 4 A.M., during which he consumed much liquor and smoked excessively. A similar one occurred New Year's day with a party continuing to 2:30 A.M., and the enjoyment of his son's wedding the following day. The directions to stop tobacco, and lose weight (now 15 pounds), and to play golf were followed. A fiery man, who screamed over the telephone and broke his golf clubs when he got a poor shot, he now admits that he merely says "Dear, Dear" when he slices into the rough. However, his business pressures are still heavy, he has suffered the collapse of a secondary investment venture in Vermont, and he is in the midst of financing a new experimental invention with a turmoil of banks, SEC registration, and stockholders meetings. He could perhaps represent the behavior pattern of the tense executive alleged to be susceptible to coronary disease, but he is mesomorphic, and was overweight and a heavy smoker. Stress, however, is still a factor in his life, yet he feels fine and has a normal electrocardiogram.

2. An insurance executive of 47 was first seen in 1951 in the hospital with a posterior myocardial infarct. He is an extremely hard driver in his business, having a very profitable insurance general agency. After his recovery, he returned to his strenuous competitive career. His weight, which he had reduced to 156, has risen in 9 years to 182, but from a consumption of 60 cigarettes a day he abruptly reduced to zero. This man has no interest in anything but work, which he says never tires him. He overdoes in food, liquor, business, and long trips. He adds more work, opens new companies, and insists on having two Cadillacs, runs a variable mild hypertension, has gout and polycythemia, and gets very little exercise. He has grieved at the loss of a grandchild and worries about his wife's hysterectomy and her continual complaint that he is antisocial, but he has no symptoms. What has protected him for 9 years from the dire effects of stress?

3. Twelve years ago I saw a 46-year-old sales engineer for a large machinery manufacturing firm and was uncertain about the cause of his bizarre chest pain, but 2 years later he had a severe myocardial infarct. A tense situation had existed in the office and he had felt better when on the road inspecting the engineering work. He had been a two-pack-a-day smoker, and liquor had at times been victori-
ous. After recovery it was thought wise to
confine him to office work where he was ex-
posed to a boss who was addicted to the
Martini lunch pattern, and who held his
liquor poorly. The patient also hates tele-
phones and crowds and is generally frus-
trated. Four years after his attack his wife,
whom he adored, died lingeringly of meta-
static cancer of the brain following a breast
operation and craniotomy the year before. A
year later he remarried a much younger
woman who does not fit his pattern of mar-
rriage successfully. His weight originally was
165 to 170 and is now 155. He smokes a pipe,
but no cigarettes. Two years ago his 14-year-
old son was killed in a friendly tug football
game. This man appears pale and tired and
has some angina with effort, but works
steadily. After 10 years of emotional agony,
he has the electrocardiographic signs of his
old infarct, but normal blood pressure, good
heart sounds, and no cardiac enlargement.
Why does he not clot fatally?

4. Almost 11 years ago, a 49-year-old man
in the amusement business was seen by me in
an emergency at home in the throes of a severe
myocardial infarct. He had had no symptoms
until 5 days before, when he had had sub-
stantial pain during coitus. He had been under
no unusual chronic or acute stress in business
prior to the attack, and is a quiet, rather
gentle sort of man.

He made a good recovery. His weight,
which in 1952 was 160, is now 147; cigarette
smoking, which was heavy, has stopped com-
pletely. Golf, which he played rarely, he now
contrives to play most of the year, at home or
in Florida. He returned to work at full time,
but during all these years he has also been
very active in the behind-the-scenes politics
of Massachusetts, gambles at the races, has
had horses of his own, and has survived his
nervous wife's menopause. At times he has
mild angina.

In the past 3 years he has been much dis-
turbed by business, politics, and family
troubles. His daughter had a miscarriage, his
dughter-in-law left a child in a car and the
child released the brakes, allowing the car to
go over a bank; he lost $5,000 betting on his
own horse; he put a large amount of his
money into the campaign of the surprise loser
for position as Mayor of Boston; the winning
opponent has destroyed his business; and in
December 1959 his beloved daughter died in
childbirth. While some of this man's stresses
are not those peculiar to Western civilization,
I must confess that I wonder, if the emotions
are so atherogenic or thrombophilic, why this
man has had no further coronary attack in 10
years. What indeed was the cholesterol, and
clotting time, and fibrinolytic activity of Job?
We know only that he survived his tribula-
tions, regained his prosperity by Godliness,
and lived for 140 years.

It is standard operating procedure in dis-
cussions of this sort for the opposing forces
to belabor each other with references from
the literature—a sort of Battle of the Bibliog-
rphies, the weight of shot and shell being
measured by volume and caliber of the
authors. I am no exception, but shall attempt
to place a few bulls'-eyes, and not resort to
saturation bombing.

I note that Dr. Harry J. Johnson, Presi-
dent of the Life Extension Foundation, in the
August 22, 1960, issue of U. S. News and
World Report was interviewed on the subject of
"How to Work Harder and Stay Healthy." His
answers were based on an examination
by his agency of about 20,000 executives a
year. Here are a few quotations: "there is no
particular health hazard to the job of being
an executive. Any health hazard is the result
of his routine of living outside the office.''

"Quite often the healthiest fellows were
those who put in the longest days and did the
hardest work."

Question: "Did the business men in your
survey say they were tense during the day?
Did they list this as a problem?"

Answer: "'No. We were amazed again to
find in this study that there were just 13.3 per
cent of executives who stated they felt they
were under undue or constant tension; 87 per
cent stated they were under some pressure,
but it didn't bother them particularly. It was
part of their job and they rather enjoyed it.'"
I don’t believe we have any higher percentage of people suffering from excessive tension among executives than in any other group of workers.’’ “They do not have a corner on ulcers, or coronary heart disease.” The man who needs most looking after is the assistant vice-president.’’

“We’re not convinced that tension plays a very important part as a cause of heart disease. We find heart disease in people whose jobs are quite free of tension-producing situations in about the same proportion as we find in the others.’’

Moving to the other end of the spectrum of masculine motivation, we find McCullagh and Lewis reporting on a study of Trappist monks.

I am not prepared to prove anything about the emotional status of Trappist monks. Psychiatrists say they don’t even have normal safety valves. For all I know, they may grind their teeth, or clench their hands, when exposed to psychological tests, but certainly the accepted image of the monk is as noted in an editorial in the New England Journal of Medicine, [emotional tension] “should hardly be compatible with what is obviously a life of contemplation, removed from the ordinary stresses of existence.”

But the study shows that, while the Trappists have relatively low blood cholesterol levels, they have not been spared from arteriosclerosis or arterial hypertension; in fact “the data suggest that arterial hypertension is more frequent in them than in other men of the same age in the American population generally.” All this, I may add, in spite of a lifetime of “placidity, frugality, and abstinence from all animal foods, except milk and very small amounts of cheese.” They do indulge in daily exercise and the figures suggest to me that they live longer than the average.

From a report by Barnes, Ratzenhofer, and Tacherne, we learn that a review of 24,546 autopsies shows that seven times as many heart attacks occurred in Graz, Austria, in 1958 as in 1944 and 1945, and they say “it is hard to imagine more stress at any time than during the War and the occupation of Austria.” I agree, even though some implicate the unique socioeconomic stress of Western man, and gaily dismiss the stresses of flood, famine, and pestilence and war by saying that they are not significant, since these are “outside the individual’s control” and “since in all forms of group emotional stress, such as war, the individual’s stress is either submerged or lost.” What happens to individual stress when it is “submerged”?

In any event, the paper by Stewart7 that is quoted in this connection is, after all, from a British pen describing a social system toward which we are tending, but have not reached; a system that he says contains the “tattered survivors of the former ‘middle classes’”. The Chicago studies show incidence rates of coronary heart disease to be high in all strata of the middle-aged male population of the United States.8

I have referred to the genetic determinant of atherosclerosis and it is evident in some degree in familial hyperlipemic states. It may be some fault in enzyme systems possibly linked with a peculiar somatic or psychic pattern. Fortunately, we are all heterozygous for most of our genetic inheritance, making us, as higher mammals, very difficult to quantitate. In pure strains of mice, in which the history of the inheritance may approximate 1,000 years of human life, certain traits may emerge. For example, in two such strains the individuals of one race, when given the choice of a beverage of pure water or dilute alcohol, will invariably choose the alcohol. Were one to suppose compulsive drinking of this sort, in the absence of the knowledge of racial influence, to be based on emotional stress, one might attempt a complex psychological testing to explain it. The reason these mice drink alcohol is that it is offered to them. Similarly, I believe that the human male develops atherosclerosis, or coagulation-fibrinolysin imbalance, if the genetic factor is present and the environment of diet, tobacco, physical inertia, plus long hours of work, or other things, perhaps forced on him by socioeconomic stress, are operative. As Dr. Stewart Wolf9

Circulation, Volume XXIII, May 1961
EMOTIONAL FACTORS IN CORONARY HEART DISEASE

It may be that the mechanics that govern the concentration of serum lipid are brought into play in the face of demand for effort that is figurative as well as literal. The possibility that any such changes are actually related to the mechanisms of coronary atherosclerosis is entirely speculative.

Coronary disease is ubiquitous. It is too nonspecific in its targets to be the result of a single cause. Indeed, it appears to be present wherever the livers of a population are in good order. The Chicago study of a utility company shows "virtually no sub-group differences in serum cholesterol levels of white males" on the basis of socioeconomic growth. Raffle, in England, in a study of coronary disease in bus drivers and the population generally, reports, "the epidemiological evidence that stress is a factor in disease is, at best, tenuous."

It is fair to ask why I am disturbed about the categorical attribution of coronary disease and thrombosis to emotional factors. I have a few reasons:

In the first place, it is so easy and popular. The American public is constantly fed with anxiety about its stresses. It is encouraged to demand security from all sorts of worry. The result is that certain occupations are becoming classified as stressful, and therefore special legal perquisites are demanded as compensation for being a fireman, a policeman, or a politician. Doctors go into court and testify that it was obvious this or that man's job was the real cause of his coronary thrombosis. Laws are passed which inhibit the employment of older workers, and lawyers have a field day. It seems to me that this is evidence of decadence. It certainly is not in the pioneering tradition. But I suppose few of us can be pioneers nowadays.

Secondly, the tendency to consider emotional stress as confined to socioeconomic tension seems to me untenable. Engel writing recently on "A Unified Concept of Health and Disease" lists the four "factors that strain current capacities of the organism."

1. Factors which injure by virtue of physical and/or chemical properties.

2. Physical factors that lead to injury when insufficient or unavailable.

[He refers here to the essential chemical elements—oxygen, water, electrolytes, etc.—that the organism must obtain from the environment (deficiency states), and the essential substances formed in the body (e.g., hormones): insufficiency states.]


Highly individual factors will determine what particular situation or process will constitute a psychological stress for any particular individual. Nonetheless, experience permits us to recognize certain categories of phenomena as being the main means whereby psychological stress may come about.

a. One is the loss of psychic objects (as represented by persons, ideals, valued possessions, job, body function and image, social roles, goals, home, country, etc.).

b. Grief or mourning represents the most familiar response.

c. Injury to the body—infliction of pain, mutilation, etc., actual or threatened, is a prominent source of psychologic stress.

d. Frustration of drives.

What I have attempted to illustrate is that, in known victims who have recovered from myocardial infarction, the continuation of stress or even its exacerbation, often does not result in an acceleration of demonstrable atherogenesis or coronary occlusion. It may of course be possible that correction of some other factor negates the effect of the emotions.

Harold Dorn has well said "To unravel the tangled skein of causation of disease by the observation of man in his environment requires the utmost persistence, a profound skepticism of the obvious, an alertness for selective factors that have produced the most readily available subjects for study, the ability to penetrate beneath the surface of the observations, a strong distrust of what is said or seen since the unchecked human memory is probably the most unreliable research instrument in existence and a willingness to spend long hours upon the development and perfection of methods of making observations.
coupled with the patience to await the occurrence of the succession of events most relevant to the hypotheses to be tested.' This latter phrase I find most wise and cautionary.

I can only hope that coronary disease will turn out to be more amenable to changes in the environment other than repeal of the Industrial Revolution or a return to the alleged mental stability and placidity of a rigid stratified society. For as Rene Dubos says,13 "To grow in the midst of dangers is the fate of the human race, because it is the law of the spirit."

References

On Permanent Patency of the Mouth of the Aorta, or Inadequacy of the Aortic Valves
By DOMINIC JOHN CORRIGAN, M.D.

One of the Physicians to the Charitable Infirmary, Jervis Street, Dublin; Lecturer on the Theory and Practice of Medicine; Consulting Physician to St. Patrick's College, Maynooth.

Diagnosis.—Inadequacy of the aortic valves may be confounded with narrowing of the mouth of the aorta, either congenital or from diseased valves, with disease of the auriculoventricular valves, with aneurism of the arch of the aorta or arteria inominata, with nervous palpitations, and with asthma . . . The visible pulsation of the arteries, arising from the arch of the aorta, which forms so striking a sign of inadequacy of the aortic valves, is wanting in narrowing of the mouth of the aorta. The pulse also is strikingly different in the two diseases. In narrowing of the aortic orifice it is small and contracted; in inadequacy of the aortic valves it is invariably full and swelling. In narrowing of the aortic orifice there is generally a marked contrast between the pulse and the impulse of the heart. The pulse is small and contracted; the impulse of the heart is strong and energetic.
Emotional Factors in Coronary Heart Disease
HOWARD B. SPRAGUE

Circulation. 1961;23:648-654
doi: 10.1161/01.CIR.23.5.648
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1961 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/23/5/648.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/