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Cardiovascular Rehabilitation

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THE BLAKISTON DIVISION

McGraw-Hill Book Company, Inc.

New York Toronto London 1957

CARDIOVASCULAR REHABILITATION

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Library of Congress Catalog Card Number: 57-10919

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Preface

The idea of organizing a conference on cardiovascular rehabilitation evolved from discussions among physicians interested in cardiovascular disease and rehabilitation and the problems resulting from the increasing incidence of cardiovascular diseases in an aging population. The primary questions under consideration were:

1. What are the major problems facing the practicing physician in the management and rehabilitation of his patients with cardiovascular disease?
2. What are the major areas of current and future research in or related to cardiovascular rehabilitation?
3. What is required in the education of physicians, co-professional personnel, and the public in order to derive maximum benefit from our present resources and skills in cardiovascular rehabilitation?

In an attempt to answer these questions, a group of physicians participated in an informal conference that extended over a two-day period. The proceedings of a major portion of the meeting were stenotyped and are presented in this volume after editing. The chapter on research is a composite of views expressed at the meeting and in correspondence which followed the conference. For uniformity, these are combined in the form presented.

THE EDITORS

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1

Emotions and the Person with Cardiovascular Disease

Dr. White: Dr. Lee, would you please open the discussion on the psychosomatic implications for the person with cardiovascular disease?

Dr. Lee: Clinical observations of the effects of emotional stimuli on the cardiovascular system have been made since Galen (A.D. 130-200), when physicians noted the influence of the mind on the pulse. The advent of the twentieth century brought many technical advances in methodology, and with the development of sphygmomanometry, electrocardiography, x-rays, etc., it was natural that these be used in the study of the cardiovascular responses to emotional stress.

The cardiovascular responses which occur as a result of emotional stimuli may be classified as (1) those involving cardiac rate and rhythm; (2) those involving the vessels to localized areas, such as the skin, brain, heart, or kidney; (3) those involving the circulation as a whole; and (4) those affecting the physical characteristics of the blood.

The changes of cardiac rate or rhythm which occur with emotional stimuli may be any of the following: sinus bradycardia or tachycardia, sinus arrhythmia, atrial flutter or fibrillation, nodal

rhythm, ventricular premature beats, ventricular tachycardia, and possibly ventricular fibrillation.

There are a variety of changes in the vascular supply to localized areas, resulting from emotional stress. In the cutaneous arterioles, capillaries, or venules, vasoconstriction or dilatation may occur, and these are manifest by pallor, blushing, and urticaria. Raynaud's phenomenon and the initial phase of vascular headaches of the migraine type are the result of emotionally induced arteriolar constriction. Renal blood flow has been shown to be reduced by emotional stress, particularly when the stress results in systemic hypertension.

The general circulatory responses to emotional stress were studied extensively in animals by Walter Cannon. The cardiovascular changes observed included tachycardia, elevation of blood pressure, decreased peripheral resistance, visceral vasoconstriction, increased blood viscosity, and diminished blood coagulation time. These changes were similar to those which followed sympathetic nerve stimulation or the injection of epinephrine. In addition it was observed that stimulation resulted in discharge of epinephrine from the adrenal gland. This reaction was described as the response of "flight or fight."

Similar changes have been observed in man by a number of investigators. Wolff and his associates at New York Hospital have characterized these responses as follows:

1. Hypodynamic reaction pattern—a fall in peripheral resistance, no change or decreased cardiac output, occasionally bradycardia and collapse.
2. High output pattern—increased pulse rate and cardiac output, decreased peripheral resistance and perhaps a slight rise in blood pressure. This pattern is also observed after exercise or epinephrine injection and is similar to Cannon's "flight or fight" response.
3. High resistance pattern—increased blood pressure and increased peripheral resistance with little change in heart rate and often a decreased cardiac output. Norepinephrine produces similar changes following subcutaneous injection in man.

Most observers have noted these reactions as a nonspecific response to anxiety, with frequent occurrence of a mixed type of response rather than one which clearly fits one of the patterns described. Wolff and his group have observed the high output pattern primarily when anxiety and conflict were overt and easily recognized, while the high resistance pattern predominated under circumstances of repressed anger, resentment, or anxiety.

The effects of emotional stress on persons with organic cardiovascular disease have been described many times since Heberden, in the classical description of angina pectoris, noted “. . . it will come by any disturbance of the mind.” Rage and anxiety are the two emotional responses most likely to precipitate angina pectoris, but even happiness has been implicated on occasion.

In a study of patients with congestive heart failure admitted to the Cincinnati General Hospital, Chambers and his associates concluded that emotional reactions, particularly rejection, frustration, and rage, were important factors in the precipitation of congestive failure in 19 of 25 patients. Engel believes that emotional depression may be an important factor in the precipitation of heart failure. The fact that emotional depression may result in altered renal function with retention of sodium and water lends support to this impression.

Some evidence has recently been presented by Ecker and associates that “strokes” may be precipitated by emotional stress.

This phenomenon has been observed clinically. As an example, a patient who displayed peculiar behavior at work for about a month finally was discharged as a result of aberrant conduct. Within 5 minutes of the time he was discharged the patient developed permanent hemiplegia. The mechanism by which this reaction occurs is unknown, although Ecker, using the technique of cerebral angiography, has postulated it is due to cerebral vascular spasm.

It is difficult to discuss emotional stress and cardiovascular disease without consideration of hypertensive cardiovascular disease. There is no definite evidence that this is an emotionally induced disease, but it seems clear that emotional stress may exacerbate the hypertension and the symptoms of the disease.

The symptoms actually are those of anxiety and bear little relation to the level of blood pressure or the cause of the disease. Psychiatric management may relieve the symptoms but has no apparent effect on the natural history of the disease.

It appears that any patient with organic cardiovascular disease may demonstrate symptoms of the disease as a result of emotional stress. In some the symptoms are the result of the physiological alterations in cardiovascular function which have been described; in others they cannot be attributable to this but are the result of as yet undisclosed mechanisms.

Dr. White: Dr. Fisher, would you care to comment?

Dr. Fisher: As a continuation of Dr. Lee's remarks, there are two questions that arise: One is, how are emotions translated into physiological responses? Second, is there any definite evidence that the emotions can play a part in the pathogenesis of any of the cardiovascular diseases?

As to the first question, there are only two systemic channels through which this can occur. One is through the nervous system, and the other is through the endocrine system. In the field of neurophysiology, I shall briefly mention some of the recent advances: (1) the localization of areas in the cerebral cortex which exert a control over the autonomic nervous system; (2) the development of the understanding of mechanisms of the ascending reticular system of the brain stem and the relationship of this system to the problems of consciousness (since this system operates in the brain stem, where so many areas are connected with the function of the autonomic nervous system, it is apparent that any future developments in this area will have a bearing in establishing a link between perception, emotions, and physiological response); (3) the evolution of clearer understanding of the rhinencephalon (the so-called "limbic system"). There has been a great deal of work in this field, and I mention this for greater perspective of this problem. In the field of neurophysiology there have been extremely important advances in the past 10 years which promise a great deal for the future, both for psychiatry and somatic medicine.

In the field of endocrinology there are (1) the development by Selye of the adaptation theory, (2) the advances made as a result of the discovery of cortisone, (3) the recent clearer understanding of salt and water metabolism following the isolation of aldosterone, and (4) the improved biochemical techniques for the measurement of blood and urine levels of epinephrine and norepinephrine in human subjects.

As to the question of pathogenesis, to my knowledge there is no definitive work which establishes to any degree of satisfaction a connection between emotion and the cause of any of the cardiovascular diseases. There are individual case reports, such as the one which Dr. Lee mentioned, and there have been many publications on psychoanalytical studies, in which a particular personality pattern or a specific emotional conflict has been labeled as the cause of hypertension, coronary occlusion, or cerebrovascular accidents. I think it can safely be stated that there is no clean-cut evidence which demonstrates that emotions cause these diseases.

Dr. White: I would like to mention the conclusions which I have reached in a very small number of patients who have heart disease due to a functional cause. It has been mentioned that nervous tension and psychological reactions do not produce heart disease, but there is a small group of persons who have arrhythmias that are easily induced, though seemingly not originated, by neuropsychiatric disturbances. Arrhythmias may be attended by tachycardia for long intervals of time, as in the case of thyrotoxic patients before adequate treatment. I started my experience long enough ago to have seen thyrotoxic heart disease patients with uncontrolled thyrotoxicosis develop fibrillation and tachycardia, cardiac enlargement, and failure. This was due in part, at least, to the tachycardia and perhaps to other influences.

Another type, also rare, may be the occurrence of paroxysmal tachycardia of one type or another recurring frequently for years because of excessive nervous stimulation.

Not infrequently, patients with varying degrees of coronary atherosclerosis will behave differently. In addition, with the same degree of atherosclerosis some patients will survive and others

die with an attack of angina pectoris, even though they do not have advanced atherosclerosis. Others who have a much higher degree of atherosclerosis can survive many attacks. This is not based wholly on the extent of collateral circulation developed. There is a change in rhythm, either cardiac standstill or ventricular fibrillation, perhaps induced either by physical strain or by psychic influence. Sudden deaths may occur in individuals who hardly seem to have enough coronary insufficiency to warrant it. There have been reports of very rare cases of deaths due to fright in otherwise healthy persons.

I wonder if Dr. Levy would comment?

Dr. Levy: Of course, Dr. White, we know that many individuals die with advanced atheromata of the coronary vessels who have never had any symptoms. Others may have just one small attack and die suddenly. There is much we do not know with regard to basic causes. As a consequence, effective treatment and rehabilitation are difficult to carry out.

In applying any form of therapy, whether it be medical, physical, or psychiatric, we must recognize that in many forms of heart disease we are dealing, not with a fixed condition, but with a dynamic one which undergoes many variations. This is particularly true in coronary disease and rheumatic fever, since in these there may be either progression or regression.

Dr. Hellerstein: While it is nice to introduce the emotional reasons why some people die suddenly and others do not, a recent study indicated that 50 per cent of the patients who die of coronary artery disease die in cardiac standstill, while the remainder die in ventricular fibrillation.

Interestingly enough, those who die in either standstill or ventricular fibrillation can be resuscitated. Although the percentage is as yet unknown, ventricular fibrillation after occlusion can be defibrillated. A physician who had just completed rounds at the University Hospital in Cleveland suddenly dropped dead. He was carried to the emergency room, about one hundred feet away, and instead of dying with his boots on, he was resuscitated with his clothes on. Through an immediate chest wall incision his heart

was defibrillated and a posterior wall infarction found. He survived. Similarly patients on the medical wards who develop ventricular fibrillation can also be resuscitated. I believe the most important part of rehabilitation is to have a live patient to rehabilitate.

Dr. White: I would like to add another method of resuscitation. Once I revived a patient who was apparently dead from a coronary occlusion by applying an ampule of aromatic ammonia to the nose. That is a pretty strong stimulant. That patient is still living years later. The heart as well as the respiration had stopped before application of the ammonia.

Dr. McDevitt, would you say a word about peripheral vascular disease?

Dr. McDevitt: Some years ago we were much interested in the effects of suggestion on the response to therapeutic procedures. I remember one man with thromboangiitis obliterans who, when he entered the study, could only walk pain-free one-quarter of a block. After 6 weeks of treatment with wet towels and a machine that would light up but had no physical therapeutic effect, he was able to walk 40 blocks without leg pain. At this point we discontinued the study, because he was beginning to develop angina pectoris.

We also observed an interesting reaction in a woman with Raynaud's phenomenon. On exposure to cold as low as 2 degrees below zero nothing happened. People were beginning to doubt the diagnosis, until someone mentioned inadvertently that her husband was an invalid whom she had taken care of for many years. Immediately her peripheral vessels went into spasm, and she developed the classical signs of this disorder.

Dr. White: Dr. Sprague, would you comment?

Dr. Sprague: There is just one point upon which I would like to comment. We must recognize the extraordinary specificity of angina at times. One may see an individual who can watch a boxing match on television without precipitating angina but who

will develop angina if he watches a wrestling bout. Perhaps he projects himself into the role of the wrestler and not the boxer. One wonders if some of these variations in the trigger mechanism may not have psychic causation that we do not recognize.

Dr. White: One of the important aspects of the problem is that of attempting to determine by methods, or techniques, the effects of emotion on circulation. Dr. Dock, who has used the ballistocardiograph a good deal, may want to comment on this or other methods of study.

Dr. Dock: In the last issue of the *Annals of Internal Medicine*, there is a very interesting report by Dr. Thomas in Baltimore, who studied the medical students at Johns Hopkins with reference to family histories. Particular interest was given to the smoking response. Boys and girls, and not as many girls as boys, whose parents had hypertension tended to respond to smoking with a greater rise in blood pressure than a control group of students whose parents were not hypertensive. The children of people who had coronary disease without hypertension tended to respond to smoking with decrease in cardiac output as shown by ballistocardiographic curves. It is very striking that one group had children responding in one way and the other group had children with the reciprocal response.

In hypertensives, some people think this has something to do with the wiring diagram which you inherit. But most of us would not have thought that a tendency to develop coronary occlusion would be associated with a decrease in cardiac output on smoking. Dr. Thomas's material will have to be carefully studied, because these are average figures. We have been interested in the response to smoking, and we would like to know if the decrease in averages was due to striking changes in a few subjects. In our student body at State University of New York we have many students, with bad family histories, manifesting either hypertension or myocardial infarction, and we have detected early asymptomatic infarction in boys under the age of twenty-five. There are extremely bizarre responses to smoking in their ballistocardiographs. These are boys whose cholesterol patterns

were abnormal, so that what these studies indicate is precocious disease, not a tendency to later development.

There is always a problem in evaluating any test procedure. A smoking test also involves an emotional response in the individual while he is smoking. We do not know what we are studying when a person smokes. To one person this may have quite a different significance than for another, so every one of these smoking tests is involved with emotional response.

The subject knows his response to smoking is being studied, and this may have an influence on physiological mechanisms. Dr. Wolff's people used ballistocardiographs some years ago and found that generally on Monday cardiac outputs were low, reached a peak on Wednesday and Thursday, and then began to fall off. There is an apparent week-end decline in the vital powers that is very striking, and the emotional interviews did change ballistocardiographic responses as well as systolic and diastolic pressures. This appears to be well recognized.

Dr. White: Would you want to comment on any other methods, for example, the use of the cardiac catheter?

Dr. Dock: The more you do to the patient before you test him, the less useful it is from the standpoint of effective psychosomatic influences. By the time the patient has had a catheter run down into his heart, his mood is disturbed by the procedure. Even putting him in a room where there is a gadget changes his mood. We all have seen simple-minded patients greatly improved by having an electrocardiogram taken. This was especially true in the old days when he had the buckets and foot basin. The patient went into the room with these gadgets with the wires in sight, was tested, and a few days later in the clinic he would say, "Couldn't you send me back to have another treatment?" The test itself affects the patient in all of the methods of study.

Dr. White: It is a very good idea to have that on the record. You don't suppose we could have any superimposed test in addition to the effects of what is being done, for instance, in the case of cardiac catheterization?